

**ABSTRACT**  
**OF**  
**The Proceedings of the Thirty-Seventh**  
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**of Life Insurance Medical**  
**Directors of America**

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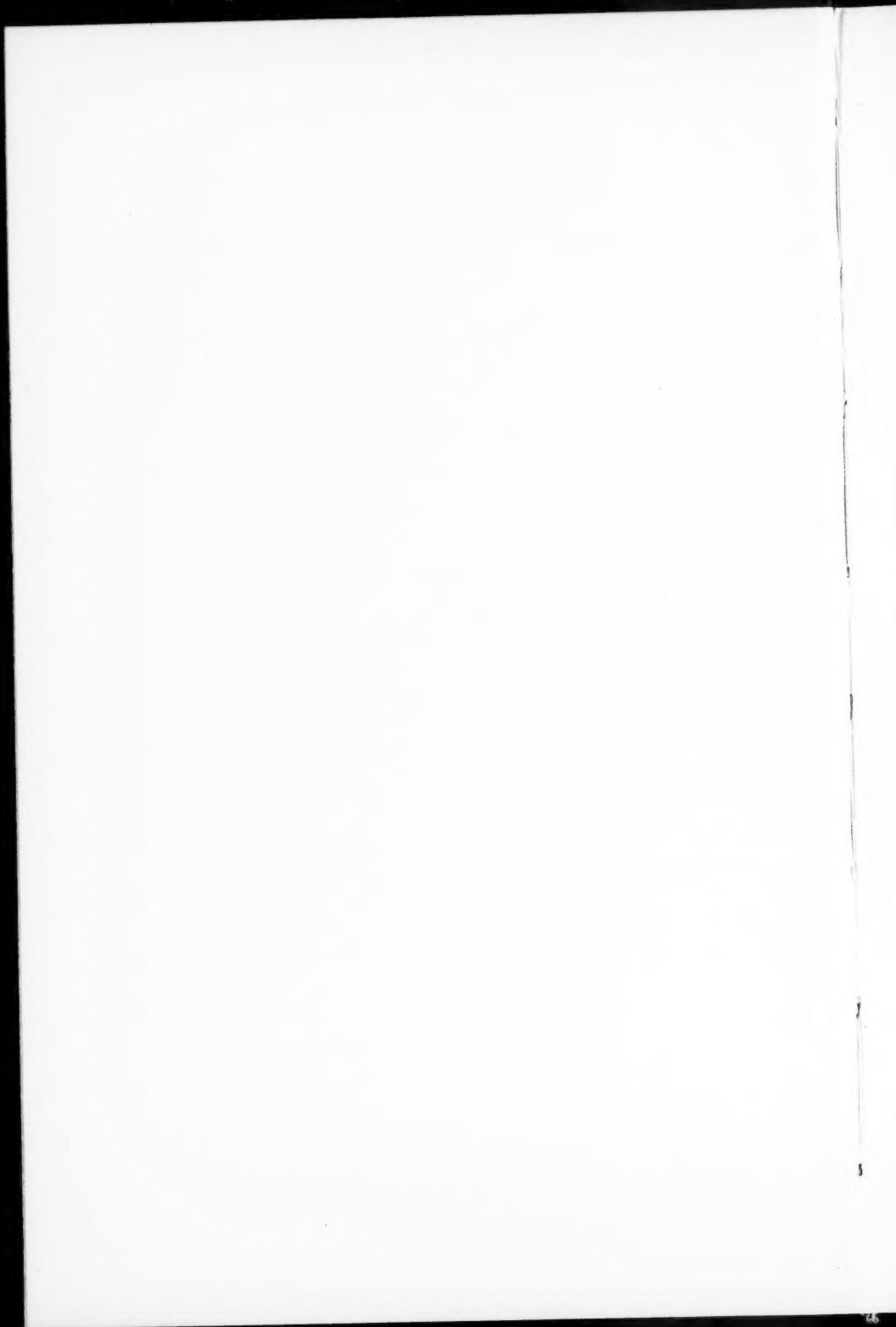
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THE ASSOCIATION OF LIFE INSURANCE  
MEDICAL DIRECTORS OF AMERICA



Compiled by the Editor of the Proceedings  
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An Abstract of the Proceedings  
OF THE  
Association of  
Life Insurance Medical Directors  
of America

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THIRTY-SEVENTH ANNUAL MEETING

The Thirty-seventh Annual Meeting of the Association of Life Insurance Medical Directors of America was held at the Waldorf-Astoria Hotel, New York City, on October 21st and 22nd, 1926. The Association was the guest of the New York Life Insurance Company of New York. President Angier B. Hobbs was in the chair.

The following members and delegates were present at some time during the sessions: Angier B. Hobbs, Eugene F. Russell, H. A. Baker, W. L. Mann, Thomas H. Willard, John S. Phelps, W. F. Milroy, S. W. Gadd, W. W. Beckett, S. C. Stanton, Jas. T. Montgomery, Wm. B. Aten, J. F. Honsberger, Wm. Muhlberg, C. M. Whicher, Joseph E. Pollard, Faneuil S. Weisse, Walter A. Jaquith, Donald B. Cragin, J. E. Kinney, Fred L. Wells, E. E. Wishard, DeWitt Smith, J. K. Gordon, Edward K. Root, Euen Van Kleeck, F. L. Grosvenor, Robert M. Daley, W. H. E. Wehner, Joyce T. Sheridan, J. L. Siner, A. E. Johann, Ross Huston, W. G. Hutchinson, O. M. Eakins, Henry R. Carstens, F. A. Causey,

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G. E. Crawford, Milton T. McCarty, J. B. Steele, Frank L. Truitt, J. Allen Patton, C. N. McCloud, Martin I. Olsen, Harry M. Brandel, LeRoy C. Grau, Carl Stutsman, Edwin W. Dwight, Harry Toulmin, Chas. B. Irwin, F. L. B. Jenney, C. F. S. Whitney, H. M. Finnerud, H. Crawford Scadding, S. J. Streight, Harold M. Frost, Daniel M. Shewbrooks, R. L. Rowley, W. E. Thornton, David N. Blakely, Edwin H. Allen, William B. Bartlett, C. H. English, C. F. Martin, T. W. Burrows, Edward McP. Armstrong, Henry W. Cook, Carleton B. McCulloch, B. F. Byrd, J. W. Amessee, R. L. Willis, L. G. Sykes, H. W. Gibbons, Ernest M. Henderson, F. W. Rolph, J. P. Turner, C. E. Schilling, A. Graham, E. G. Dewis, S. B. Scholz, Robert J. Graves, T. F. McMahon, J. M. Smith, Henry Colt, R. W. Mann, A. S. Knight, B. Y. Jaudon, Frank Harnden, T. D. Archibald, W. F. Blackford, H. W. Dingman, H. L. Mann, John L. Adams, Robert J. Kissock, J. B. Hall, M. C. Wilson, Herbert Old, M. B. Bender, Charles B. Piper, Oscar H. Rogers, Paul Fitzgerald, F. W. Chapin, T. H. Rockwell, W. G. Exton, J. T. J. Battle, J. W. Fisher, Gordon Wilson, C. L. Rudasill, Morton Snow, A. O. Jimenis, Arthur H. Griswold, J. H. North, W. M. Bradshaw, F. W. Hagney, F. W. McSorley, Lefferts Hutton, J. T. Cabaniss, Wm. S. Gardner, Paul Mazzuri, Henry A. Bancel, Edwin B. Wilson, William R. Ward, F. I. Ganot, W. P. Lamb, H. M. Decker, C. A. Van Dervoort, H. W. Billings, G. Holbrook Barber, G. E. Kanouse, Thayer A. Smith.

On motion the roll call was waived and the members were requested to register their names in the book provided for that purpose.

On motion the reading of the minutes of the thirty-sixth annual meeting of the Association, held on October 22nd and 23rd, 1925, was waived.



## Election of New Members

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The names of the following candidates recommended by the Executive Council for membership in the Association were presented:

- Dr. Dewitt Smith, Southwestern Life Insurance Company, Dallas, Texas.
- Dr. John C. Young, American Life Insurance Company, Detroit, Michigan.
- Dr. Joseph C. Horan, The Metropolitan Life Insurance Company, New York City.
- Dr. Henry L. Mann, The Equitable Life Assurance Society, New York City.
- Dr. G. E. Allen, National Life Assurance Company, Montpelier, Vermont.
- Dr. J. Charles Humphreys, Penn Mutual Life Insurance Company, Philadelphia, Pa.
- Dr. Joyce T. Sheridan, Philadelphia Life Insurance Company, Philadelphia, Pa.
- Dr. Edwin B. Wilson, Mutual Life Insurance Company of New York, New York City.
- Dr. Washington C. Huyler, Mutual Life Insurance Company of New York, New York City.
- Dr. W. L. Mann, The Great-West Life Assurance Company, Winnipeg, Man., Canada.
- Dr. Wade Wright, The Metropolitan Life Insurance Company, New York City.
- Dr. Anthony J. Lanza, Metropolitan Life Insurance Company, New York City.

On motion, duly seconded and carried, the Secretary was instructed to cast a ballot in favor of the election of each of these candidates.

The Secretary announced the ballot so cast, and the candidates were declared elected to membership in the Association. The President appointed Drs. Toulmin and Muhlberg

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as a Committee to introduce the newly elected members to the Association. Only two of these members were present, Drs. Sheridan and Mann, and they were escorted into the room by the Committee.

The Secretary read the list of delegates who were elected to attend the meeting from the American Life Convention, as follows:

Dr. H. M. Decker, Register Life, Davenport, Iowa.  
Dr. M. T. McCarty, Peoples' Life, Frankfort, Iowa.  
Dr. H. M. Brandel, Great Republic Life, Los Angeles, Calif.  
Dr. F. N. Cochems, American Life, Denver, Colo.  
Dr. G. E. Crawford, Cedar Rapids Life, Cedar Rapids, Iowa.  
Dr. J. E. Kinney, Farmers Life, Denver, Colo.  
Dr. H. M. Finnerud, Midland National, Watertown, S. D.  
Dr. B. F. Byrd, National Life & Accident Ins. Co., Nashville, Tenn.  
Dr. H. R. Carstons, Detroit Life Insurance Company, Detroit, Mich.

The Secretary read the minutes of the meetings of the Executive Council of May 19 and 20, 1926. On motion these minutes were adopted as read.

Dr. Hobbs—And now it devolves upon me to say that our membership has not been intact, during the past year. There are certain members who have been with us before that are now missing, and I will read the names of those deceased members:

Dr. Donald M. Gedge, Metropolitan Life Insurance Company, New York City.  
Dr. James T. Priestley, Royal Union Mutual Life Insurance Company, Des Moines, Iowa.  
Dr. Robert L. Lounsberry, Security Mutual Life Insurance Company, Binghamton, New York.  
Dr. John Mason Little, New England Mutual, Boston, Mass.

## Memorial of Dr. Gedge

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The following memorials were read and on motion the Secretary was instructed to have them printed in the Annual Proceedings and to send copies of the memorials to the families of the deceased members.

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DONALD MACCOLLOCH GEDGE, M. D.

(Presented by Dr. Thomas H. Willard)

We are called upon to note the passing of one of the members of this organization who by reason of his location on the West Coast of the country was seldom an attendant at our meetings, but was an interested and valuable member.

Donald MacColloch Gedge was a graduate of the Cooper Medical College in San Francisco in the class of 1892. In 1901 he became a field medical examiner for the Company in the City of San Francisco, and in 1906 he was appointed Assistant Medical Director of the Metropolitan Life Insurance Company, which position he held until the time of his death. His taking away was sudden, although it was known for some time that he had not been in his usual robust health. He had presided at a reunion of his Medical College class and a half an hour after the conclusion of the session was taken alarmingly ill and in a few minutes expired in the arms of his wife.

His early life had been a very interesting one. He had spent much time in the far east—Cochin-China and in the South Seas. He had relatives in Honolulu and his wife was born there. His memories of his adventures and his word painting of the scenery of the South Seas were interesting and enthusiastic. He was, as he probably might have described himself, "a bit of a poet" and some of his friends have among their cherished possessions a slender volume of his effusions, which revealed much of his sweetness of character and love of beauty.

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He leaves a widow, a daughter and two grandchildren to mourn his loss, and it is with great sincerity that those who knew him will feel keenly the loss of one who was a good physician, a faithful Officer of his Company, a sincere friend and a loving companion.

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### DR. JAMES TAGGART PRIESTLEY

1852-1925

(Presented by Dr. Charles M. Whicher)

The medical fraternity lost one of its most illustrious sons, the Royal Union Life Insurance Company lost a brilliant Medical Director, and the State of Iowa suffered the loss of a beloved pioneer in the death December 11, 1925, of Dr. James Taggart Priestley.

Dr. Priestley was born in Northumberland, Pa., July 19, 1852. He was descendant from Scotch and English ancestry and was the great-great grandson of the historically famous chemist, Dr. Joseph Priestley, the discoverer of oxygen.

He was graduated from the medical college of the University of Pennsylvania in 1872, at the age of twenty years, and two years later began his medical practice in Iowa.

In March, 1886, he became the Medical Director for the Royal Union Mutual Life Insurance Company, and for nearly forty years served the Medical Department of that Company and its successor, the Royal Union Life Insurance Company, for which he was Associate Medical Director at the time of his death.

Dr. Priestley's professional life was long and busy. He entered into the medical life of his city, state and nation with great interest. He served as President of the Iowa State Medical Association in 1900, and was honored repeatedly

by the American Medical Association, in which he served as Secretary of the Medical Section in 1896-1897, and from 1894 to 1900 as a member of the Board of Trustees. He served as Surgeon General of Iowa under four Governors.

He was a physician of rare culture and refinement and loved by all who knew him. An event which indicated the esteem of his fellow citizens was the celebration of Dr. Priestley's seventieth birthday, July 19, 1922, by one hundred and fifty local business and professional men, who gathered to pay homage to the physician. The guests included many of the doctor's associates in the work of the Iowa State Medical Association and the American Medical Association.

The climax of Dr. Priestley's long list of honors came when, in 1924, the degree of Doctor of Science was conferred upon him by his Alma Mater, the University of Pennsylvania, for his distinguished service in medicine. It is said to have been the only time in the history of the University that an Honorary Degree has been conferred in absentia, the doctor at that time being unable to make the trip to Philadelphia.

Among the notable tributes paid to Dr. Priestley was that of Drs. William and Charles H. Mayo, who said: "Dr. Priestley was a great physician of the Ian MacLaren type; a sagacious practitioner of medicine, with an almost instinctive grasp of the essential details of a case and with the human touch of a kindly sympathetic soul. For more than forty years a leader in medicine, he retained to the last his youthful state of mind, which enabled him to appreciate researches which led to advancement in his beloved profession. His life was given to a career of usefulness to humanity not excelled by any man of his time. Let us hope that condition of modern medical education and practice may enable us to develop other men of his rare qualities in the years to come."

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Dr. Priestley is survived by his widow and by two grandsons, Dr. Joseph Little Priestley and James Taggart Priestley, 2nd.

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### DR. ROBERT LANNING LOUNSBERRY

1869-1926

(Presented by Dr. William B. Aton.)

Dr. Robert L. Lounsberry, a member of the Board of Directors and Medical Director of the Security Mutual Life Insurance Company of Binghamton, New York, died suddenly of angina in his berth on the train near Adams, Wis., early in the morning of July 8, 1926, while returning from an Agency Convention held at Minneapolis, Minn. His death was a very distinct shock to all of his associates, friends and family. He was apparently in robust health; no one ever having heard him complain in any way.

Dr. Lounsberry was born February 14, 1869, in Tioga County, New York, and came of a pioneer family, his grandfather having settled in this community in 1804, his father being born on the same farm. He was educated in the public schools in Tioga County, Wyoming Seminary, and received his medical degree from New York University in 1889. He practiced medicine for two years in Owego, New York, removing to Buffalo in 1892, where he practiced medicine until 1896 when he became associated with this company as Assistant Medical Director. In December, 1897, he was made Medical Director of the Company upon the death of Dr. Edwards. Had he lived until December, 1926, he would have completed thirty years of service with this company.

Dr. Lounsberry was a man of remarkable personality. He was possessed of rare judgment and a highly stored mind. A

## Memorials of Dr. Lounsberry and Dr. Little 9

never-failing fund of humor was his, along with the faculty of being able to illustrate almost any given point with a relevant story. He was a man who hid a great kindness of heart beneath a bluff exterior, the welfare of the humblest employee being as much his concern as that of any official of the company.

In the fullness of a rich life Dr. Lounsberry has answered the call of the Great Beyond. Security Mutual has suffered a real loss, the loss of a Director, a valued official, a wise counselor and a true friend. His memory, fragrant with many kind deeds and works, will still be with us as the years go by. He, in truth, gave his life in the service of his Company.

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JOHN MASON LITTLE, M. D.

1875-1926

(Presented by Dr. David N. Blakely)

Dr. John Mason Little died at his home in Brookline, March 23, 1926, of heart disease after an illness of two weeks. He was born in Swampscott, Mass., June 7, 1875.

He was a graduate of Harvard College 1897 and Harvard Medical School 1901.

After serving an internship at the Massachusetts General Hospital he spent a year in travel and study in Europe. He returned to Boston in 1903 and began private practice.

In 1907 he joined the Grenfell Mission and for ten years was very active in its work in Newfoundland and Labrador. During most of this time he was the Head of a small hospital at St. Anthony, Newfoundland.

In September, 1911, at St. Anthony, he married a co-

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worker, Miss Ruth Esther Keese, of Ashburnham, Mass., who survives him, together with five sons and a daughter. In 1917, for the sake of their children, Dr. and Mrs. Little reluctantly returned to Massachusetts.

After his return he was appointed surgeon to out-patients at the Massachusetts General Hospital, Assistant Visiting Surgeon to the Long Island Hospital, Instructor in Surgical Technique at the Harvard Medical School and a Medical Examiner of the New England Mutual Life Insurance Company. Later he became one of the Assistant Medical Directors of that Company and Chief Surgeon of the Boston and Albany Railroad. This necessitated his resigning his hospital and medical school positions.

He was elected to membership in this Association in 1922. Although he gave but part time to insurance work the many-sided problems of medical selection aroused his keen interest and his associates valued his wise counsel based on his unusually broad experience. He contributed a chapter on "The Abdomen" to the book, "Life Insurance Examination," edited by Dr. F. W. Foxworthy and published by the C. V. Mosby Company in 1924.

He was also a member of the Massachusetts Medical Society, American Medical Association, American College of Surgeons, the Harvard Club and the Union Club of Boston.

After graduation from college in 1897, ten years were spent in medical school, hospital, graduate study in Vienna and private practice, under the inspiring leadership of the late Dr. Samuel Mixter, whose assistant he was and whose surgical practice took him to many parts of New England in the days when means of transportation were less rapid than at present and when well-equipped small hospitals were far less numerous than now. Frequent operations on patients in their homes tended to develop the resourcefulness of the surgeon. Then came ten years of the varied work, medical, surgical



and administrative, of the well-known Grenfell Mission. This period presented professional and sociological problems numerous enough and complex enough to tax the ingenuity and test the courage of any man or woman who tried to meet and dispose of them. The short summers were spent in cruising up and down the coast of that North Country and in the winter many trips were made by dog sled. His letters to his family, written with no thought of publication, reveal experiences as full of thrills and of dangers as those of the arctic explorers. As to his medical and surgical problems, no ordinary description can convey an adequate idea of either their multitude, their magnitude or their variety. One must read and reread the letters trying to picture the background of isolation, arctic climate and primitive conditions generally, including ignorance and poverty. One should remember, too, that such isolation means that the surgeon must be not only physically but also professionally bold and resourceful, for there are no consultants available and often no assistants; he alone stands between his patient and continued suffering or even death. During the years of this difficult and absorbing work Dr. Little was eminently successful. He published several articles in medical journals, notable among which was a report of a series of operations for the relief of selected cases of epilepsy and a summary of his studies of beri-beri as seen in that country. During the last ten-year period of his life, after consideration of his family had brought him back to his native state, he continued his keen interest in the welfare of the people "Up North." At the time of his death he was President of the New England Grenfell Association and a Director of the International Grenfell Association. Membership in these bodies was not an honorary position merely, but one which involved much hard work and called for faith, tact and courage, all of which were given cheerfully and freely.

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A many-sided man who made friends easily, who knew how to play but who seemed to prefer hard work, who gave generously of time and counsel to his friends, a man of broad vision and sympathy, yet well-poised and with a lively sense of humor, such was the friend we have known, and whom we shall hold in grateful and affectionate remembrance.

Dr. Angier B. Hobbs, President of the Association, delivered the following introductory remarks to his address:

This is the Thirty-seventh Annual Meeting of our Association—an association with a history of accomplishments and without doubt a future full of possibilities. A year ago, when you elected me President, there came a desire to see the organization made more influential, more widely known and of greater practical value to its members and the companies they represent. No definite plan, I regret to say, has suggested itself as to how such a desirable result can be brought about. Perhaps an additional publication, issued as opportunity seems to require, might prove a feasible project. Such a publication could contain contributed articles of general and varied interests without necessarily a background of statistical study. In addition to this, our Publicity Committee might put the Association on record as endorsing the work being done by public spirited organizations endeavoring to raise the standards of health, sanitation, education and to foster proper research, experimentation and investigation. The By-laws seem to give this Committee full and discretionary powers to do this.

Aside from any visionary schemes, we can all agree that our proceedings should reflect not only progress along familiar lines but also live ideas in connection with newer activities. Life Insurance Companies today are not satisfied with old objectives but are seeking ways and means to include in their policy contracts additional benefits available to the insured. This is now evidenced in the effort to cover

disability, partial or total, due to disease or injury. While statistics may be lacking regarding these new phases of the business, is it not possible to have a more general interchange of opinions, experiences and practices which will be of assistance in avoiding mistakes and obtaining more uniform action? Every day we are facing questions arising out of more liberal practices, many of them unforeseen, unprovided for and eventually solved only through legal routes. A publication supplementary to our proceedings would furnish the means of accomplishing this end.

In regard to our regular meetings, when we consider that our membership is well trained in an interesting and specialized field of endeavor and, in the larger companies at least that there is a wide experience to draw upon, it should be easy to form a program suitable to our yearly gatherings. It has not worked out this way. It is not at all difficult to choose appropriate subjects but quite so to find those willing to contribute articles on assigned topics. The usual plea for declining to enter the lists is the lack of statistical material. Such is, no doubt, often a valid excuse and I take this opportunity, therefore, to express appreciation and thanks to those who have consented to serve us in spite of this and other obstacles. Some of the contributions this year are shorter than had been expected but this will have the advantage of leaving a larger margin of time for profitable discussion and for the consideration perhaps of topics not included in our program.

My own contribution is Syphilis; a Study. The further I investigated this subject, the more I was impressed with its importance and the wide-spread occurrence of the infection. The last ten or fifteen years have been replete with acquired knowledge of the disease and the time seems opportune for bringing our statistical study up to date. It would have been desirable to draw some conclusions respecting old and new

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methods of treatment but this was impossible. The old was merged into the new with no distinct dividing line apparent in our records.

### SYPHILIS; A STUDY.

BY ANGIER B. HOBBS, M. A., M. D.

*Medical Director, New York Life Insurance Co.*

The last time syphilis was under any special consideration by this Association was in January, 1919, when a symposium was held on the subject, with four of our members contributing to the study. Since that time we have learned much about this disease—so much that we can almost say our knowledge is fairly complete and dependable. When we recognize the universality of specific infection, its innumerable complications and its possible after effects on all organs of the body, we cannot but realize its importance to those of us vitally interested in the selection of risks.

The history of syphilography, while interesting, is but of little practical value. There are some facts, however, connected with an up-to-date knowledge of the disease itself, which it may be worth while to consider in a condensed review, compiled from recent expositions of the subject.

No reliable figures are available as to the prevalence of syphilis but we can form some general conclusions of more or less value. Osler says, "Syphilis is the despair of statisticians." That perhaps absolves our actuaries, but, if so, where can we turn for a basis of medical selection. One-fourth of the deaths in the first year of life have been attributed to syphilis and those that are syphilitic and survive are frequently subject to innumerable maladies which give us trouble later in life as insurance risks. The U. S. Census reports more than ten thousand deaths every year due to syphilis, including tabes and paresis. When we include the

various disorders produced by syphilis, the total number of deaths due to this disease may conservatively be said to be over 25,000 annually.

Among the diseases attributable to syphilis are paresis, tabes and a certain proportion of apoplexy, epilepsy, encephalitis, arterio-sclerosis, heart disease and nephritis. There are also a large number of suicides among these people.

In many of our hospitals Wassermans are done as a routine and from 20 to 30 per cent give a positive reaction. Greeley (Mon. Bulletin, Dept. Health, New York City, March, 1915) states that 22 to 25 per cent of all patients admitted to Bellevue Hospital give a positive reaction. In considering these figures, however, we must have in mind the class of patients admitted to a general hospital. A member of our Home Office staff was at one time connected with a prominent and well equipped hospital in the middle west, where the class of patients was above that to be found in general city institutions. He tells me that 10 per cent of all cases admitted (practically none on account of a syphilitic infection) showed a positive Wasserman. Vidder of the U. S. Army concluded that 20 per cent of men enlisting showed a syphilitic infection. In general, authorities tell us that 10 to 15 per cent of the population of large centres are syphilitic. The percentage of syphilitics is lower among women than among men and higher in the negro than in the white race.

As to the infection, syphilis is either acquired (in a few cases perhaps innocently) or congenital. The discovery of the organism causing syphilis was the beginning of the scientific study of the subject. This was in 1905 and made by Schaudinn and Hoffmann, who gave it the name *Spirocheta pallida* (a protozoon), later known as *Treponema pallidum*. The latter name was adopted by the International Committee on Nomenclature, which makes it the preferable term. Since that time this organism has been intensively studied and been found

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in all of the lesions of syphilis in every stage of its development. It is found in greater numbers in primary and secondary lesions than in tertiary lesions. It is found abundantly in active congenital syphilis. When treponemata gain entrance to the body through mucous membrane or abrasion they multiply rapidly, especially in the immediate neighborhood of the locality of infection. The deep lymphatics are involved early but the treponemata have not become established in distant parts and can be more readily reached by intensive treatment. It is thus easily understood why early diagnosis is most valuable.

*Course of the Disease:* The period of incubation has been placed at 10 to 60 days—average about 21 days. During this period, while there are no symptoms, the causative organisms are increasing in numbers sufficient to produce an inflammatory lesion—the chancre. This is the earliest phenomenon of syphilis—the primary stage.

The possibility of syphilis being acquired without the exhibition of a primary lesion has long been recognized but most of these cases are probably instances where the primary sore has escaped notice, as for example, where the source of infection lies in the urethra. Such cases are not as rare as some suppose and are important to us as explaining those that exhibit symptoms which can be considered syphilitic and yet give no history of a primary stage.

Some authorities speak only of an early and late stage of syphilis. The more convenient classification, however, is the time honored one of primary, secondary and tertiary.

The primary stage is the chancre. Symptoms of the secondary stage are well known but may be so slight as to escape notice. Even in the first stage the chancre may be only a pimple and remain unobserved. Rarely in untreated, frequently in treated cases, secondary symptoms may fail to appear.

After the secondary stage is the period of latency when the treponemata are dormant and the patient in good health with no sign of the disease except perhaps a positive Wasserman. This period usually lasts two years, although the interval may be from a few months to thirty or more years.

Following this is the tertiary stage, where the characteristic lesion is the gumma, a nodular neoplasm, a cellular proliferation with a tendency to fibrosis, small, large or even diffuse from which no tissue of the body seems to be exempt. While the lesions of the tertiary stage are distinctive and vital in character, they do not inevitably appear, some say in only fifteen per cent. Adequate treatment may prevent later symptoms and even in untreated cases they may be absent.

*Congenital Syphilis:* The term congenital is preferable to hereditary because the infection is transmitted to the foetus in utero. Congenital syphilis and the acquired form have the same characteristics, except there is no primary stage in the former and it is apt to be more intense. Symptoms of congenital syphilis are often absent at birth, in fact, infants later found to be syphilitic are apparently healthy at this time. Osler states that 75% of children with history of congenital syphilis die. Available figures seem to be quite uncertain but it is apparently a fact that only a small proportion survive childhood and early youth. Hazen says that 3% of all syphilitics seen in private practice are affected with the congenital form—in dispensary practice, 8% to 10%. This is only his own experience but it leads us to conclude that an appreciable number of all syphilitics are congenitally syphilitic. Later or delayed congenital syphilis, appearing at puberty or in after life, presents a varied group of diseases. The nervous system is very apt to suffer—paralysis, epilepsy, tabes, paresis and a predisposition to organic and functional neuroses. Next in frequency to diseases of the nervous system are gummata of the skin and viscera. Some form of valvular

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disease of the heart and aortic aneurysm are due to congenital syphilis. Liver, spleen and kidneys are at times the seat of cirrhotic changes, the result of this infection.

As to the Wasserman Test in congenital syphilis, it may be stated in general that a positive reaction may be expected in retarded cases with more frequency than in the acquired form.

*Diagnosis:* The number of syphilitics in the U. S., and I suppose this includes all with syphilitic manifestations, has been estimated to be from six millions upwards. The diagnosis of this condition is, therefore, of serious moment. We have two methods of diagnosis, the clinical and the laboratory and when we consider that there are many indisputable evidences of syphilis in a clinical history, we must hesitate to endorse laboratory findings as always conclusive. At the same time the serological test, known as the Wasserman reaction, with careful interpretation, has been one of the greatest contributions to the diagnosis of this disease. While there are other tests requiring the most exact technique, this is the one universally used. The results of the Wasserman reaction vary during different stages of the disease, the highest number of positive reactions being obtained in the secondary stage, the lowest in this order, primary, latent and tertiary.

When the diagnosis of chancre itself is in doubt, the demonstration of the presence of the *Treponema pallidum*, in a drop of serum from the chancre by means of dark field illumination with exact technique, is conclusive. Some authorities say that as high as 80% of such cases can now be diagnosed.

*Nomenclature of Wasserman Results:* All laboratories do not use the same nomenclature. In many a four plus (++++) indicates a complete inhibition of hemolysis. Hemolysis will not occur if the patient is syphilitic. Three plus (+++), two plus (++) and plus (+) indicate



lesser degrees of inhibition. The Army uses double plus ( $=4$  plus), plus ( $=3+$  and  $2+$ ) and plus minus, the last being the same as one plus of other laboratories.

**Interpretation of Wasserman Results:** It may be said in general that a plus or plus minus Wasserman or a negative Wasserman—alone—cannot be taken as a basis for a positive or negative diagnosis. Many factors may render a Wasserman reaction negative when it should be positive. Only negative reactions persisting over a long period of time, some say a year, should be taken as evidence of the absence of the disease and even then the spinal fluid should be tested.

Other diseases being excluded, a plus four reaction may be considered as diagnostic. A three plus or two plus reaction in primary, tertiary or latent infections may be so considered if there is a history of infection or of clinical symptoms. A plus or even a two plus reaction alone, without history, should not be the basis for positive diagnosis—nor should a negative reaction except as corroborative evidence.

It has been proven that the ingestion of considerable amounts of alcohol will change a positive Wasserman to negative. The Wasserman reaction shows positive in trypanosomic diseases and at times in tuberculous leprosy, febrile malaria, carcinoma, sarcoma, pseudoleukaemia and rarely in other conditions even when syphilis has been excluded. The practice of lumbar puncture and the withdrawal of cerebro-spinal fluid for examination is an important aid to differential diagnosis. The blood may be and often is negative in nervous syphilis but not as a rule the cerebro-spinal fluid. This will usually be positive.

**Treatment of Syphilis:** Syphilis is not a disease calling for drug experimentation in treatment. We know of three specifics, arsenic, mercury and potassium iodide, ranking in importance as named. The first two are used in curing the disease—the last, having no effect on the *Treponema pallidum*,

will cause an absorption or elimination of its morbid products. Arsenic is the most powerful of these three drugs—the form used being known as Arsphenamine, Salvarsan or 606. It was invented by Ehrlich and represented his 606th trial experiment. Arsphenamine contains 31+ % of arsenic. Neo-arsphenamine or Neo-salvarsan contains a lesser amount of arsenic but is more simple and preferable for administration, remembering its potency is about  $\frac{3}{5}$  that of Salvarsan. These are Treponemacidal fluids and careful technique must be observed in their use as it involves some contraindications and dangers. The second drug of the trio is mercury—successfully used in combating the infection of syphilis for centuries and not to be entirely discarded now for arsenic, despite the opinions of a few clinicians.

Potassium iodide. The value of this drug increases with the age of the disease and its potency is directed most successfully against the exudates appearing later in the infection and against the lesions of congenital syphilis in conjunction with mercury.

*Thorough Treatment:* In regard to constitutional treatment, authorities differ as to details only. The scientific subcommittee of the Medical Society of the State of New York sponsors the following plan of Dr. Walter M. Burnet and Dr. Albert Pfeiffer: "The plan of treatment most frequently followed is this: One course of arsphenamine or sulpharsphenamine intravenously, 6-10 doses, at weekly intervals; after the first week of abortive treatment one course of mercury intravenously 10-20 doses, intramuscularly 10-30 injections or by inunction 15-40 rubs. Some such plan of treatment should cover about three months and then a blood test. If negative less intensive treatment for eight weeks and a Wasserman. If blood is still negative, treatment of some kind should be continued. Wassermans should be made at monthly intervals for a number of months and every three

months for two years from date of primary lesion. A spinal fluid examination should be made on all patients before being discharged as cured or arrested. More intensive or prolonged treatment is necessary in the cases which show positive blood reactions after one or more courses of arsphenamine and mercury." Some authorities advise a spinal fluid test at the outset in late cases.

I think we should be impressed with the necessity of some such thorough treatment with proper tests before we consider our applicants as cured.

*After Effects:* Late syphilis is responsible for a large percentage of deaths among insured lives and a fair number of disability claims. When we have a known or doubtful history of this infection we can, if careful in our selection, provide against the future. There will always, however, be a large number of cases where the disease is congenital or its occurrence unacknowledged or unknown. First and foremost among diseases having a syphilitic basis are those of the nervous system, especially paresis and locomotor ataxia.

*Paresis:* In nearly all cases the cause is syphilis with often a modifying factor, especially alcohol. While there are instances of apparent cure or at least arrest—probably one-half the cases die within two years and four-fifths within five years. The disease makes its appearance in from five to fifteen years or more after the initial lesion. It is said that one or two per cent (some authorities give a higher percentage) of all syphilitics develop paresis—more common among men than women.

*Locomotor Ataxia:* With hardly a reservation this disease is due to syphilitic infection. Its frequency may be one-half of that of general paresis. Lucke, studying 250 cases of his own and using other statistics, found an average period of beginning to be about 15 years after the primary sore. It is known that in tabes, as in other tertiary nervous lesions, the

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interval between infection and the appearance of the disease shortens with age. When epilepsy develops after the age of 30 to 35, syphilis should be suspected. There are many of these cases—aura usually absent and treatment more or less effective.

The part that syphilis plays in other disorders of the cerebro-spinal system has not been as yet thoroughly investigated and conclusions may be incorrectly drawn.

Other important after effects of syphilis, from an insurance standpoint, relate to the cardio-vascular system. It is especially known to attack the walls of blood vessels. Cabot in his recently published work, "Facts on the Heart," reports 92 autopsies of syphilitic aortitis. In 41 of them the disease produced aneurysm—in 26 aortic regurgitation—in 3 an associated aortic stenosis—in 22 the change represented a latent condition. Syphilis while a very common cause of aortic regurgitation, does not play the same part in connection with other valvular diseases. In the former instance, it is the part of the picture of aortitis, as is also disease of the coronary arteries with the symptoms of angina pectoris. The pericardium and myocardium are at times the seat of syphilitic changes.

*Prognosis in Syphilis:* In the last generation Dr. Hayes Agnew said that the man who has syphilis will have it all his life and his ghost will have it when he is dead. Fournier said, "Syphilis does not die, it only sleeps." These opinions perhaps expressed an extreme view but were in a measure justified when mercury and potassium iodide were the only means to combat the disease. This picture has in some degree changed with our present knowledge and the result has been brought about by the discovery of the *Treponema pallidum*, the Wasserman test and Arsphenamine—all in fifteen years. These facts furnish the basis for the assertion that our knowledge is now fairly dependable.

Even the manifestations of syphilis seem to have changed

and for reasons unknown. We now have less of the gross cutaneous and osseous lesions and more of the visceral lesions and those of the nervous and circulatory symptoms. In the main, it may be said that whoever has a positive Wasserman reaction (there are some exceptions), has syphilis and unless we have changed the positive to a negative reaction, we have not cured syphilis. Further, we must be convinced that the negative Wasserman is permanent. It must be emphasized that the conclusion of cure cannot rest on two, three or even more Wassermans, unless repeated at intervals for a considerable length of time. While modern methods enable us to cure or eliminate syphilis, the treatment must be thorough and the cure confirmed. The outlook is not as serious as some of the after effects might lead us to believe and the mortality of "cured" syphilitics can be fairly estimated, as our statistics show.

#### MORTALITY AMONG PERSONS WITH A HISTORY OF SYPHILIS

In Volume IV of the Medico-Actuarial Mortality Investigation, published in 1914, the experience of a number of companies is given among persons with a history of syphilis. These cases were all accepted as standard risks. In the group of those thoroughly treated, with one year's freedom from symptoms, the ratio of actual to expected deaths by the M. A. Select Table was 188%. In the group of persons not thoroughly treated, or who did not give the details of treatment, the relative mortality was 174%. In another group, in which it was doubtful whether or not the insured had had syphilis, the mortality was 138%. In the report of the Committee it was stated that one company which did a large business in sub-standard lives gave its relative mortality among syphilitics as 135%. That company was the New York Life. Its favorable mortality, compared with that of the companies contributing to the M. A. Experience, may have been due, in part

at least, to the fact that its business had been more recently issued but it must also have been caused by the great care which had been exercised in the selection of these risks.

During a meeting of this Association in January, 1919, there was a symposium on the subject of syphilis, already referred to, in the course of which the Penn Mutual showed a mortality of 145% of the M. A. Table at ages of entry under 40, and 301% at ages of entry 40 and over and, as a result of this experience, they had ceased to grant insurance to applicants presenting a history of syphilis; the Travelers gave no details of their experience at that time but stated that, as far back as 1914, they had discontinued granting them insurance; the Equitable reported their experience to have been 160% of the M. A. Table; and the Mutual theirs as 153% on Life and Limited Payment Plans and 130% on Endowment Plans, combining all cases including those in which there was a doubtful history of syphilis. In the experience of the Mutual Life, the mortality among doubtful cases was higher than among those that were surely syphilitic.

By way of supplementing this testimony and bringing our information upon the subject down to the present time, I am permitted to present here the experience of the New York Life on issues of 1896 to 1924, inclusive, carried to the anniversaries in 1925. During this period all of our cases 4,490 in number, have been insured as substandard risks, because the Company has at no time been willing to accept, at the regular rate of premium, any person, however favorable his case, who has had syphilis.

The mortality has been studied by policies and the expected deaths calculated by the A. M. Select Table. This Table by no means represents fairly the mortality of the Company during the 29 years covered by this study. During the earlier years of this period, the Company's mortality was distinctly higher than the Table. Recently it has become decid-

edly lower than the Table. It is, therefore, extremely difficult to make an exact comparison of the mortality among these syphilitics and that among standard lives of the Company, especially as we must take into account the distribution according to the years of issue. But as this comparison is our main consideration in this study, this relation, as nearly as we could determine it, appears in separate ratios in the last column of each of the Tables here submitted.

For the greater part this is a study of syphilis uncomplicated by any other impairments but cases with minor impairments have been added to swell the numbers. These were not a large percentage of the total and the impairments unimportant so that the effect upon the mortality ratios here presented was slight, certainly not as much as five points.

I. The first group to be studied was that of 2,397 persons who had certainly had syphilis, who had been thoroughly treated (two years' continuous treatment) and had had one year's freedom from symptoms. No subdivision was made with regard to number of years since the incident, as by so doing the size of each group would have been rendered too small. In the following synopsis appear the results of our investigation:

Syphilis Surely, Thoroughly Treated, Two Years' Continuous Treatment and One Year's Freedom From Symptoms. Number of Cases Entering into the Investigation—2,397.

Policy Years	Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths by A. M. Select Table	Ratio (Approximate) to Company's Mortality on Standard Lives
1-5	65	48.4	134%	150%
6th & over	70	61.9	113%	122%
All years	135	110.3	122%	135%
Ages at Issue				
15-39	69	58.4	118%	132%
40 & over	66	51.9	127%	137%
All ages	135	110.3	122%	135%

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The mortality shown in this table is much more favorable than that of the M. A. Investigation and also more favorable than most of the later experiences, to which reference has already been made. It is, however, in agreement with the latter, when the fact is taken into account, that our group was very carefully selected.

The difference in the mortality ratios for the first five and for the succeeding policy years does not indicate any serious selection against the Company. A division of the data by ages at issue did not show that there was much difference in the relative mortality by ages.

Of the 135 deaths, 21 were directly attributable to syphilis, such as paresis, locomotor ataxia, etc. There were 10 from heart disease and 8 from apoplexy. Twenty-four died from malignant tumors, an unusually high percentage.

We had hoped to be able to determine the effect of the recent improved treatment of the disease but found that it was not feasible to do so for the reason that in too many instances the facts of the nature of the treatment were not given in sufficient detail. The mortality in recent years has been distinctly better among them than in the earlier years covered by this investigation but the mortality among standard risks has also greatly improved during the same period. Taking account of this improvement among standard risks, the mortality among syphilitics has been reduced and the more recent treatment of syphilis is undoubtedly more effective than the old, but I hesitate to make an estimate of the extent.

II. Another group, made up of 272 persons, was taken with the same history as the foregoing and under the same conditions except that there was a history of tuberculosis in the family. The material is not large enough to justify subdivisions.



Syphilis Surely, Thoroughly Treated, Two Years' Continuous Treatment and One Year's Freedom From Symptoms, With a Family History of Tuberculosis. Number of Cases Entering into Investigation—272.

Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths by A. M. Select Table	Ratio (Approximate) to Company's Mortality on Standard Lives
27	16.2	167%	185%

This indicates a higher mortality, as would have been expected. Of the 27 deaths 7 were due to paresis. Otherwise there was no significant feature in the causes of death.

III. If, in addition to a history of syphilis, there was a record of excesses or of the rather free use of alcohol, the risks were selected with greater care and on higher ratings. Cases of this sort, 174 in number, yielded the following:

Syphilis Surely, Thoroughly Treated, Two Years' Continuous Treatment and One Year's Freedom From Symptoms, With an Occasional Alcoholic Excess, or Where Insured Drank Freely but Not to Excess.

Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths by A. M. Select Table	Ratio (Approximate) to Company's Mortality on Standard Lives
19	8.7	218%	235%

Among the 19 deaths there were none from paresis, locomotor ataxia and other diseases directly attributable to syphilis but there was a very heavy death rate from heart disease, cirrhosis of the liver and suicide.

IV. Another group, 990 cases, was analyzed of those who had syphilis but the treatment was not thorough or full information regarding the treatment could not be obtained. A synopsis of the results was as follows:

Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths by A. M. Select Table	Ratio (Approximate) to Company's Mortality on Standard Lives
44	33.6	131%	145%

The mortality of this group is in harmony with that of the first group where the treatment was thorough. Of the 44

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deaths, 6 were from locomotor ataxia or other diseases directly attributable to syphilis and 6 were from apoplexy.

V. The last group, 657 cases, were those in which it was doubtful whether the insured had had an attack of syphilis or not. The synopsis of this part of the study is as follows:

Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths by A. M. Select Table	Ratio (Approximate) to Company's Mortality on Standard Lives
48	32.2	149%	165%

The foregoing experience was unexpected as it shows a higher mortality than in the cases where the insured had had syphilis. It may be due to three reasons:

(1) That there was a tendency to throw cases into this group if full information could not be obtained.

(2) The agents, knowing that more liberal treatment was given to those in the doubtful group, may have induced us to put insured into this group who were not entitled to be so placed.

(3) If it were doubtful whether the applicant had the disease or not, proper precautions may not have been taken and medical advice not followed.

The third is probably the most likely explanation for the higher mortality than expected in this group. A study of the death losses shows that probably as large a proportion had the disease as in the "Surely" group. Of the 48 deaths, 10 were from diseases directly attributable to syphilis, such as paresis; and 11 died from heart disease, some of which may very well have been due to syphilis.

We had hoped to study the effect or build in connection with a history of syphilis, but when the data were divided into the various percentages of departure from the average weight, the material was found too small to be of any value. We, therefore, grouped together all cases in which the ab-

dominal girth was more than two inches greater than the chest at rest, which would represent the persons definitely overweight. There were 18 deaths in this group, with expected deaths of 7.7—a ratio of 230% of the A. M. Table. It might be inferred that overweight with a history of syphilis is not a good combination. Of the 18 deaths 5 were from cerebral hemorrhage and apoplexy, 4 from Bright's disease and 2 from heart disease—a rather significant commentary on the combination of impairments.

In May, 1926, the Canadian Life Insurance Officers' Association issued Bulletin No. 45, giving recommendations regarding selection in two groups with a history of syphilitic infection:

UNDERSTANDARD—CURED, THOROUGH TREATMENT

"Cured" means more than one negative Wasserman

Initial lesion only +35

Slight secondary symptoms +50

Marked saturation +60

(Indicating virulence of infection)

Tertiary symptoms R. N. A. (Risk Not Advised)

In cases more than five years since date of attack, when there is a doubtful diagnosis of a primary lesion only, a credit of 5 to 10 points might be given for a reliable history of repeated negative tests over a term of five to twenty-five years, the last within one year.

While undergoing a course of treatment which has lasted less than two years but more than one year and which otherwise conforms with the above requirements, rate as under Understandard—Cured Through Treatment.

UNDERSTANDARD—CURED, NOT THOROUGH TREATMENT

With treatment which does not satisfy conditions but with no symptoms for at least 2 years prior to date of application.

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Initial lesion only	+75 and up
Slight secondary symptoms	+100 and up
Marked saturation	+150 and up
(Indicating virulence of infection)	
Tertiary symptoms	R. N. A.

In cases more than five years since date of attack, a credit of 10 to 25 points might be given for a reliable history of repeated negative tests over a term of five to twenty-five years, the last within one year.

The Committee desires to emphasize its opinion that risks with a definite history of Understandard should never be accepted as standard at standard rates, even on Endowment plans of insurance.

The foregoing is very valuable in connection with the selection of risks as it gives the consensus of opinion of medical directors and actuaries with an extensive experience.

### CONCLUSIONS

1. Where there is evidence satisfactory as to thorough treatment and cure and no other impairments, an average mortality rating of from 135% to 140% will probably cover in carefully selected cases. Some cases are somewhat more favorable and others less so. In my opinion none are standard risks. We cannot exclude but we do, of course, ignore unforeseen after effects.

2. In our cases partially treated only or where details of treatment were unknown, but without other impairments the ratio is increased to some extent. Here I do not believe our statistics are convincing. This is a group where the syphilis becomes latent—just enough treatment to make it so. The after effects “loom” large. I would not hesitate to say that the mortality would be somewhere over 150% or more in spite of figures given.

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3. In cases with minor but significant impairment, when the syphilis is considered cured, such as tuberculosis in the family history, the mortality is increased say to 170-185%. If we regard, as we must, a family history of tuberculosis in underweights as significant, we must realize that an additional factor tending to produce a cachexia must increase the mortality to an appreciable degree.

4. In alcoholic cases—not very excessive—the mortality is considerably increased—even among those thoroughly treated—to about 225%.

5. The longer treatment is delayed, the less liability there is of cure. For this reason the cases where secondaries have developed must receive a higher rating than those mentioned above.

6. Where tertiary symptoms have appeared, there is no chance of so-called cure and we should consider such cases as practically uninsurable.

7. We must always bear in mind the far-reaching after effects of this disease and not treat well founded suspicion too leniently.

Dr. Hobbs—We will postpone the discussion of my paper until we have finished the routine business.

The Nominating Committee presented the following report:

The Nominating Committee desires to report the following list of nominees as Officers for the ensuing year:

For President—Dr. W. W. Beckett.

For First Vice President—Dr. R. M. Daley.

For Second Vice President—Dr. J. A. Patton.

For Secretary—Dr. Chester T. Brown.

For Treasurer—Dr. Charles L. Christiernin.

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For Editor of the Proceedings—Dr. E. F. Russell.

For five members of the Executive Council—Dr. G. A. Van Wagenan, Dr. E. W. Dwight, Dr. Wm. Muhlberg, Dr. Ross Huston, Dr. Morton Snow.

All of which is respectfully submitted.

C. F. S. WHITNEY,  
ROBERT M. DALEY,  
W. R. WARD,  
HARRY TOULMIN,  
CHESTER T. BROWN,  
THOMAS H. WILLARD, Chairman.

Dr. Hobbs—You have heard the report of this committee. The by-laws provide that there can be additional nominations if you so desire. If not, I will entertain a motion that the nominations be closed.

No further nominations being presented, it was moved by Dr. Eakins and seconded by Dr. Weisse that the nominations be closed, and that the Secretary be instructed to cast a ballot for the officers and members of the Executive Council placed in nomination by the Nominating Committee. The motion was carried.

Dr. Hobbs—Dr. Christiernin, our Treasurer, is ill, not seriously, but he is unable to attend the meeting and deliver his report in person, but he has submitted it for the Auditing Committee. I would like to have the Auditing Committee, which consists of Dr. Muhlberg and Dr. Jaquith, report to-day if they can do so.

Dr. Jaquith—We will postpone the report of the Auditing Committee until the audit is completed.

Dr. O. H. Rogers, Chairman of the Special Committee in charge of the M. I. B. presented the report of that Committee, which was accepted with thanks and ordered placed on file.

Dr. Hobbs—The next committee report is on blood pressure, but I am informed by Dr. Fisher that he considers it a better place for the report of his committee at the time that the paper on blood pressure by Dr. Rogers and Mr. Hunter comes before the Association, so we will postpone the report of the Blood Pressure Committee until that time.

We will now have the report of the Committee on Public Health.

Dr. Willard—I haven't a formal report but nothing has been presented before the committee. However, in view of the fact that the recommendations of the President are that this organization as an organization shall identify itself with the work that is being done by various institutions, we do believe that it would be well to continue the committee, because one cannot tell just what moment some recommendation from the committee or some activity of it may not be induced by an absolutely unsuspected happening. Therefore, we ask that the committee be continued, reporting that nothing has been brought to its attention during the past year.

It was moved and seconded the report of progress be received and that the committee be continued.

Dr. Patton presented the report of the Committee for correlating Practices of Reporting Urinary Impairments.

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### REPORT OF THE COMMITTEE ON URINARY IMPAIRMENTS

A. Your Committee desires to report as follows concerning the microscopic methods for recording casts, blood and pus in urinalyses. These comments also cover the inquiry of Dr. D. M. Shewbrooks, which Dr. F. S. Weisse passed on to this Committee.

1. The sediment should be from about 15 c. c. or a half ounce of urine centrifuged at a rate of 2,000 to 2,500 revolutions per minute for at least 10,000 revolutions. Care should be used in applying the centrifuge brake so as not to disturb the sediment.

2. Place on a slide the entire sediment if no coverslip is to be used, or one-half the sediment if using a  $7/8$  by  $7/8$  cover-slip. Use a capillary pipette, which does not require suction, so that if using a cover-slip a representative sample of the sediment can be taken from the top, middle, and bottom.

3. The method of illumination is important. If it is some form of artificial light, the heat from this should not distort the sediment by evaporation.

4. Examine thoroughly first with low power objective  $2/3$  or 16 mm. and then as needed with high power objective,  $1/6$  or 4 mm., with due consideration to eyepiece used, so that the total magnification is known in estimating the field.

5. Casts should be reported by number per slide, but each office is to decide upon their importance or significance for itself. The number per slide will vary, based upon the use of the half or the whole of the sediment.

This Committee recommends also that the member companies discontinue reporting 1 or 2 casts, but continue the others as per 1925 code instructions.



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6. Red blood and pus and epithelial cells should be recorded separately, as so many per field. The standard field for this purpose is that of the 6x eyepiece and the 1/6 or 4 mm. objective. Enough fields should be counted (15 to 20) - to be sure the count is representative of the entire slide.

7. Red blood cells should not be reported as hematuria, unless there are more than five (5) of these cells per standard field.

8. Pus cells should not be reported as pyuria, unless there are more than ten (10) of these cells per standard field.

9. The use of symbols of multiple significance to denote the number of blood or pus cells is not recommended by this Committee.

B. Albumin reporting has been considered by this Committee and no change in official tests is deemed advisable.

The limits for amounts as per approval of this Association in 1924 at the Thirty-fifth Annual Meeting and per the rules of the official impairment codes of 1925 are believed worthy of discussion.

The table below shows the official or approved limits, the corresponding 1925 code and the limits that have been suggested for discussion.

Official	Per Cent	Mille %	1925 Code	Suggested
Small	.01 - .05	10- 50	Trace	.03 - .05 or 30-50
Moderate	.051-0.1	51-100	Moderate	.051-0.1 or 51-100
Large	Over 0.1	Over 100	Large amount	Over 0.1 or 100

A. S. KNIGHT,  
T. H. ROCKWELL,  
WM. R. WARD,

Chairman: J. ALLEN PATTON.

As there was a difference of opinion as to accepting the report, it was referred back to the committee to report next year, sending out copies of their report in advance.

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Dr. Rogers—I should very much like to be able to bring up before the M. I. B. Committee this question: Shall the committee issue a rule that hereafter the amount of albumin, when reported by the sulphosalicylic acid test, be indicated by the fraction in parenthesis? If we have the instruction of this Association to that effect, we will proceed with it at once.

It was moved, seconded and carried that Dr. Rogers should be so instructed.

Dr. Patton—For the benefit of the Urinary Committee, we should like to have each of the member companies consider this question, and if they can possibly, within the next month, send to the committee their opinions on this question of sediment, how they are going to get the casts per slide, the pus and blood cells per field, to serve as a basis for their consideration. It would be of immense help to that committee to formulate a report and get it back to you gentlemen so that you can give it consideration before the next meeting.

Dr. Rogers was asked the advisability of extending the same rule for reporting the amount of sugar.

Dr. Rogers—I don't know how widely the picric acid test has been adopted, but I think it is quite feasible.

Dr. Hobbs—I think the test for albumin is more generally used and has been more practically worked out than the test for sugar. The time may come when we can take up the sugar test and treat it the same way as albumin, but I think the albumin question is the one more to be considered at the present time.

The Treasurer being absent, the Secretary read the report of the Treasurer.

Dr. Jaquith—Dr. Muhlberg and I have audited the Treasurer's accounts and find them to be correct.

The two reports were on motion accepted and approved.

Dr. Hobbs—The next thing for consideration is the amendment to the by-laws.

Dr. Brown—The Executive Council on its meeting on October 20 approved the following by-law and recommended it to the Association for adoption:

Amendment No. 1.

There shall be a class of membership known as "Members Emeritus." Former members of the Association who have been retired by their respective companies shall be eligible. Such members shall not be assessed for dues or other fees, shall not hold office and shall have no vote. The method of election to this class of membership shall be the same as that required for regular membership.

It was moved and seconded that the above be adopted as an amendment to the by-laws.

Dr. Brown read the report of the Dreyer Committee, which had been audited, and upon motion the report was accepted and approved.

Dr. Hobbs—That finishes the routine business of the Association. The next in order is miscellaneous business. There being no miscellaneous business, we will proceed with the next part of the program, the reading of papers, and here is the point to which I postponed the discussion of the paper which I contributed on syphilis. I will call upon Dr. Weisse for his part of the discussion.

Dr. Weisse—Mr. President and Gentlemen: I have read, with much interest, Dr. Hobbs' study of syphilis and his report of the New York Life Experience.

I will recapitulate our experience for purposes of comparison, as I reported them in 1919:

NEW YORK LIFE

Issues 1896 to 1924—Exposed to 1925—A. M. Select Table

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Medical Impairment, Class 1—Entrants, 2,397; Ratio, 122%.

Classes 2 and 3 combined—Entrants, 990; Ratio, 131%.

Class 4—Entrants, 657; Ratio, 149%.

Total Entrants, 4,490; Total Deaths, 267.

### MUTUAL LIFE

Issues 1885 to 1906—Exposed to 1915—M. A. Table

Medical Impairment, Class 1—Entrants, 176; Ratio, 135%.

Classes 2 and 3 combined—Entrants, 134; Ratio, 137%.

Class 4—Entrants, 122; Ratio, 161%.

Total Entrants, 432; Total Deaths, 53.

In comparing, kindly note that the New York Life's ratio is on the A. M. Select Table, while the Mutual Life is on the M. A. Table.

As Doctor Hobbs states in his paper, our combined experience on all classes gave us a ratio of 143%; those on Life and Life Limited, 153%, and on Endowments only 130%.

Since 1906, up to and including 1922, we have issued policies on Endowment plans only to 280 individuals. This number is so small and the period of exposure is so comparatively short, that we have not worked up any figures thereon, and will probably not do so until we have had a full 20 years' exposure.

30,613 policies have been terminated by death among men insured in our Company from 1907 to 1922, exposed to the policy anniversary in 1924. Of these, syphilis, including locomotor ataxia and paresis, which are clinically known as due to syphilis, was returned as the determining cause of death in 410 cases. This figure represents 1.3% of the total deaths.

These figures are far from the truth because of the tend-

ency of physicians to conceal this disease in their reports of causes of death.

This tendency to concealment is still more apparent in the applicants for insurance, for, of the 410 cases who died of syphilis, although their average duration was 7 years, only 5 cases gave a history of syphilis.

I agree thoroughly with Dr. Hobbs that the discoveries and changes in the treatment of syphilis, which have taken place in the last 15 years, will have a definite bearing in the future on our mortality. But I do not feel that the benefits therefrom will begin to show in our general mortality for some time to come.

Dr. Daley—Mr. President and Gentlemen: While it has been definitely proven that syphilis can be cured, the difficulty of obtaining such proof only emphasizes the resistance to treatment.

A disease\* which so frequently manifests itself subjectively until irreparable harm has been done, so difficult to detect by the most careful examination, is always a source of concern to the Medical Department of a Life Insurance Company.

Syphilitic risks, accepted as such, form but a small percentage of insured syphilites, as disability and death claims so frequently demonstrate.

Dr. Hobbs has so succinctly presented the subject that little is left but to present the mortality from this disease as experienced by the Equitable Life Assurance Society of the United States, with an addition showing the effect of syphilis on the disability features of the policy contract so far as it can be determined by rough analysis. As the Equitable eliminates this clause in policies on the lives of applicants with such a history, it is evident from the character of the disabling disease that the larger part of these claims occur in policyholders whose infection predates their policies.

The mortality experience of the Equitable with syphilis risks up to 1924 has been as follows:

	Entrants	Expected Loss	Actual Loss	Ratio	M-A Experience
S. the only impairment	1200	60.30	71	118%	
S. complicated with other impairments	684	27.10	57	210%	
Total	1884	87.40	128	146%	
All S. Risks:					
1st to 5th year		36.42	45	130%	134%
6th year and later		52.78	83	157%	194%
Total		87.40	128	146%	169%

As you notice, the mortality ratios in the above tables differ only slightly from the ratios of our syphilis experience up to 1917. The mortality remains slightly higher than 100% if syphilis is the only impairment, but averages over 200% when complications have developed. The Equitable mortality to 1924 is lower than the M. A. mortality. This may be explained by the fact that the bulk of the Equitable risks in this class was taken from 1900 to 1922 and observed to 1924, while the risks for the M. A. Experience were taken from 1885 to 1908 and observed to 1909. Mortality generally has improved greatly in recent years.

Ages at Entry	Mortality by Age Groups			M-A Experience
	Expected	Actual	Ratio	
to 29	12.56	23	183%	188%
30 to 39	34.61	60	173	177
40 to 49	28.51	35	123	165
50 and over	11.72	10	86	143
	87.40	128	146	170%

The mortality by age groups shows the higher ratios at the younger ages of entry, as one might expect. Our low mortality of age group 50 and over is due no doubt to the smallness of the group, the M. A. mortality of 143% seems much more likely to be correct.

## Discussion—Syphilis

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Causes of Deaths			
Tuberculosis	8	Pulmonary dis.	13
Syphilis	3	Digestive "	9
Cancers	6	Gen. Urin. "	7
Other gen'l dis.	16	Suicides	7
Nervous "	23	Violent deaths	8
Circulatory "	23	Unknown causes	5
			<hr/> 128

The deaths from nervous and circulatory diseases and from suicide are from 2 to 3 times the normal in number.

Syphilis Disability Claims			
Total claims approved for total disability to Sept. 30, 1926	5106		
" " " " account Paresis	55 or 1.08%		
" " " " " Loco Ataxia	29 or 0.57%		
" " " " " Syphilis	27 or 0.53%		
		<hr/> 111 or 2.18%	

This is, of course, not a very reliable or exact statement, for doubtless cases of syphilitic origin are included in insanity, paralysis and even blindness.

Dr. Hobbs—I am glad to see that Dr. Daley has brought into his report a reference to disability. I think that in the coming years we are going to find that syphilis not acknowledged or admitted upon examination, perhaps unknown, is going to be a growing cause—especially in nervous diseases—for disability benefits. I think as soon as possible when material is sufficient it would be a very interesting thing to have it presented before the Association. It is a lead in the right direction. Dr. Baker.

Dr. Baker—Mr. President and Gentlemen: Our President's paper on syphilis is the best exposition of the subject from a life insurance standpoint that I have ever read. I only regret that Dr. Weisse and Dr. Daley have left so little to be said in the way of a discussion that I am practically without words on the subject.

Before we come to the point where ratings can be scientifically applied in a case of syphilis, we must first determine whether the individual has ever really had that disease. From

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the standpoint of practical underwriting here is the all-important question. A large number of cases having a supposed initial lesion fall into the hands of so-called specialists, ethical and otherwise, and energetic treatment is immediately begun without a conclusive diagnosis first having been made. Arspenamine is given in repeated doses followed by mercury and the iodides over as long a period as the patient proves a profitable one, and that man never in his life will know whether he had syphilis or not. I have no doubt that the favorable mortality in the class where thorough treatment was given after a so-called "certain" diagnosis from initial lesion only is due to the inclusion of many cases where syphilis was in fact never present. We have many cases of syphilis come before us for rating where evidently a clear history has been reported to another company, and which the applicant now denies, claiming that information was given the first company in error. It involves a nice sense of discrimination to decide the merits of such a case. We have written to attending physicians and had them reply that a positive diagnosis was made from the initial lesion only and treatment instituted in order to be on the safe side and as a matter of precaution.

From the standpoint of the patient no greater mistake could be made than to label him as a syphilitic and then remove his only chance to disprove that stigma. When we remember that a large percentage of all cases of supposed syphilis falls into the hands of advertising specialists we can appreciate the force of this argument. If treatment has been given by regular physicians in good standing it is always best to get a request for details over applicant's own signature and then usually a full report concerning the case can be secured. I believe, however, that greater attention should be paid to the symptoms upon which diagnosis was made rather than to the method and thoroughness of treatment employed. In fairness to both applicant and company all cases possible should



be selected out and given standard policies if the diagnosis is sufficiently doubtful to warrant this procedure.

Given, however, a frank case of syphilis, we always have a substandard risk. This is an axiom, and those who have insured such cases on standard forms have always had a high mortality in the class as shown by the Specialized Mortality Investigation. Dr. Hobbs' company has shown a wonderful achievement in selection in having secured so favorable a mortality in a class that is the despair of the average underwriter.

Dr. Hobbs—Dr. Rogers, will you say something, please, upon this subject?

Dr. Rogers—I am so completely in sympathy with the views expressed in the paper itself that I don't know that I can add anything. If I were to suggest anything at all, I should say that I doubt very much, even with the modern technique of treating syphilis, that syphilis is ever cured. You said in the paper, and my own view is, that syphilitics will always show a higher mortality than the normal. I doubt if a group of syphilitics can be gotten together and show a normal mortality.

Dr. Hobbs—And perhaps it is so, that from the very nature of the disease it is impossible to get an average, normal mortality out of any group of syphilitics, however good they may seem. In doubtful cases, it seems to me that companies making the selections should have the benefit of that doubt and should pass very careful judgment on past history and not be led into the error of granting standard insurance on lives where there is a great probability, at least possibility, of having a syphilitic. I think this affection is very widespread, and you will notice in the article which I contributed the percentage of syphilitics found in the various hospitals, general hospitals and private hospitals, everywhere throughout the country. It is quite a prevalent disease. While it is

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the despair of the actuaries to give us figures to get a good mortality out of it, at the same time the treatment is such today that the syphilitic has a great hope of having the disease, as far as he is concerned, practically eradicated. I do not think that there is ever a cure, as Dr. Rogers said, and I think that is the consensus of opinion. I would like to hear remarks concerning this subject from others. Has anybody else any remarks to make?

I know nothing else to give except to mention the conclusions that I have drawn in the paper which gives the experience of our company. It warns against the granting of insurance except at the highest rates where there are impairments which modify the disease, especially in regard to alcoholic and tuberculous family history. Where there are any tertiary symptoms at all, we consider uninsurable on any basis.

We will now proceed to the next number on the program with the presentation by Dr. Rogers of a new graphic table of heights and weights.

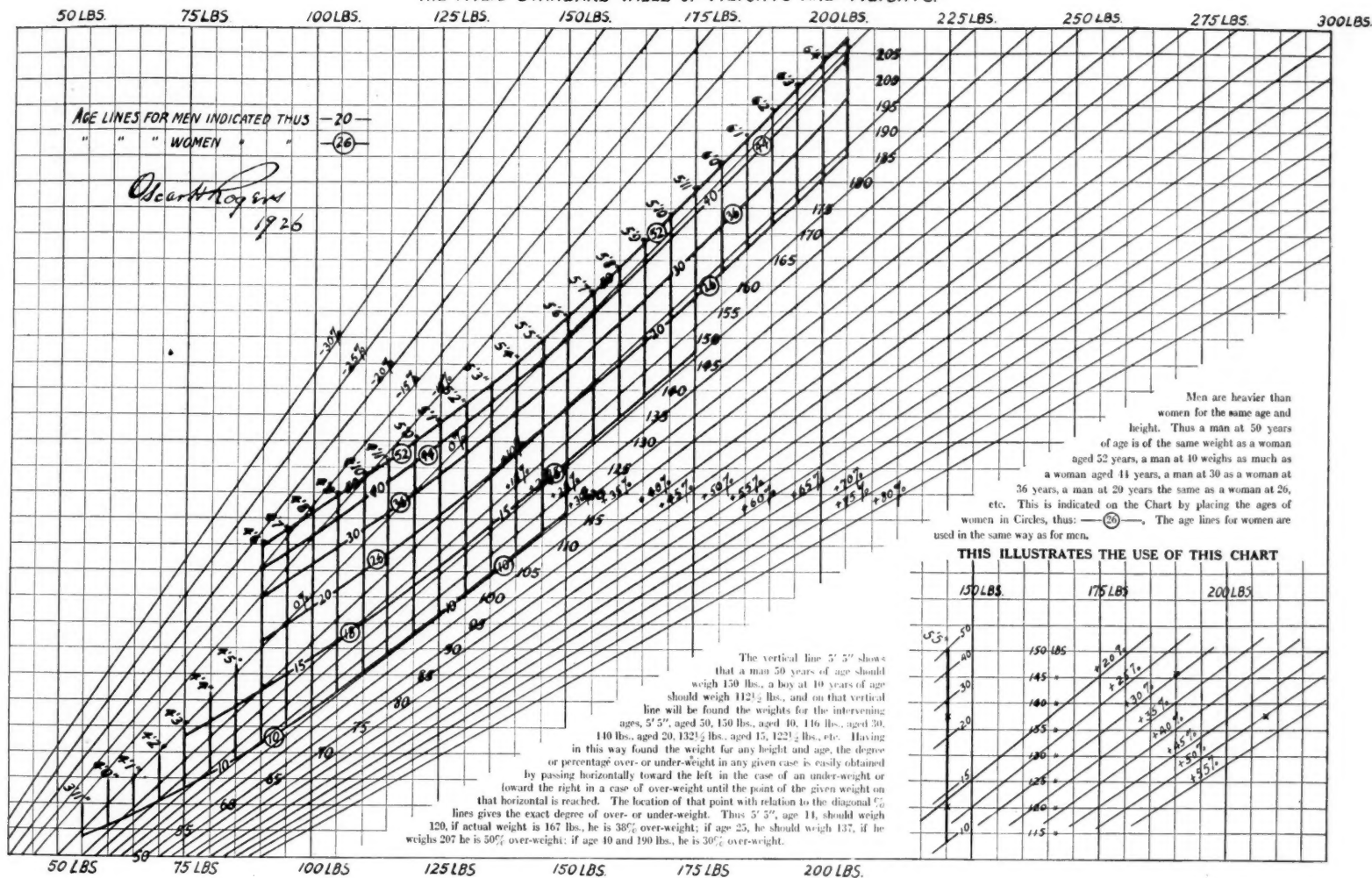
### GRAPHIC STANDARD TABLE

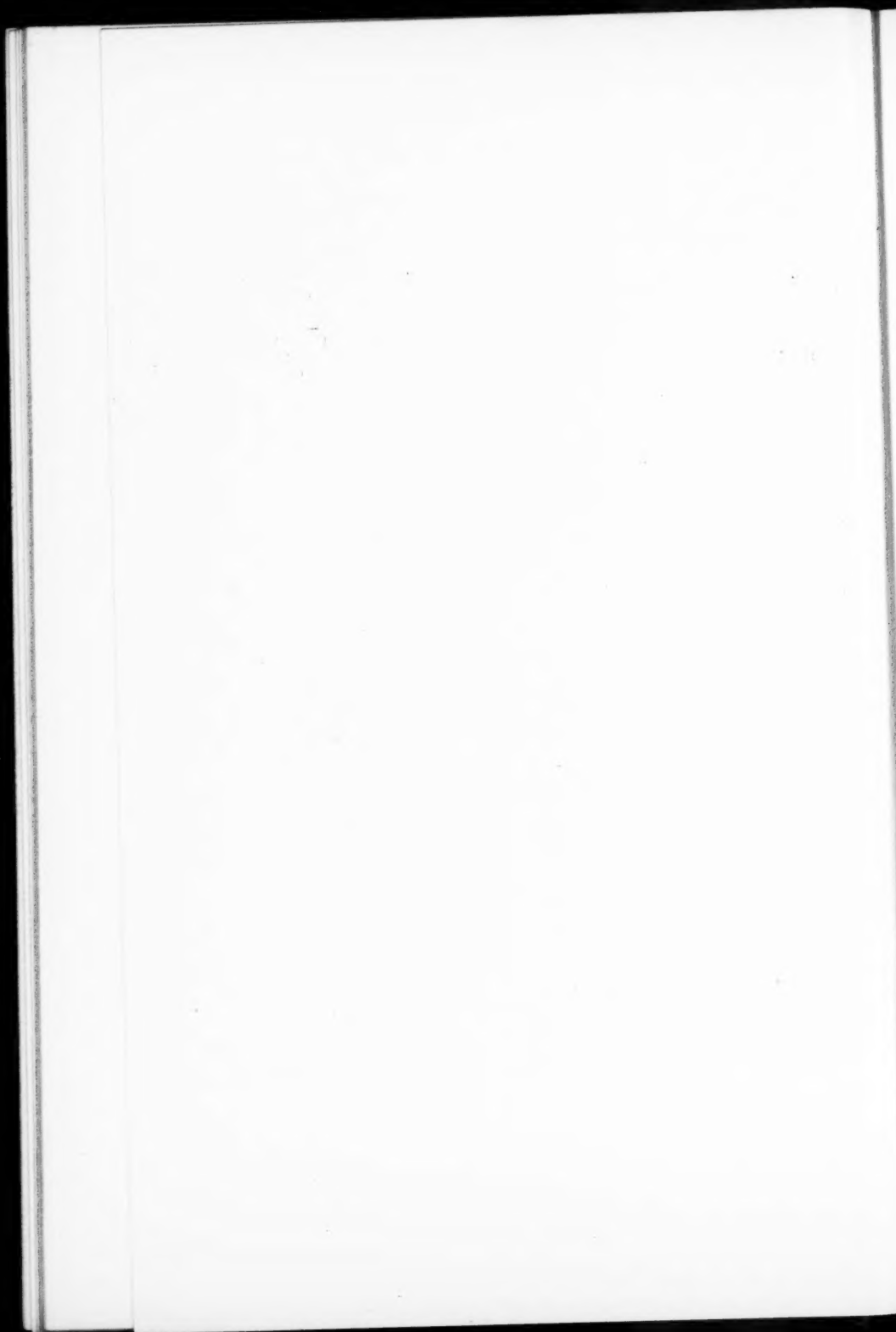
BY DR. OSCAR ROGERS

*Chief Medical Director, New York Life Insurance Company*

This Graphic Standard Table, prepared for use in the Medical Department of the New York Life Insurance Company, is the last of a series of tables which have been published from time to time since 1897. The first of the series was based upon the report of the Shepherd Committee of the Medical Directors Association. Later editions have been modified by such additional data as have been at the time available, notably, those of the National Fraternal Congress, of Dr. Weisse's Standard Weights of Women, and of the Medico-Actuarial Committee. The earlier tables dealt with

# THE NYLIC STANDARD TABLE OF HEIGHTS AND WEIGHTS.





ages 20 and over. The table of 1920 was made to include age 16. During the last few years, the benefits of life insurance have been extended to still younger persons, until now, insurance is granted to those as young as 10 years of age. In this way there has developed a real need of standards to include these younger ages.

Up to the present time, most of us have been depending, for the younger ages, upon tables of heights and weights developed outside of the life insurance business, notably, the Wood-Baldwin Table, in use in the Metropolitan Life; but this practice has never been very satisfactory for the reason that, at the time they were prepared, the standard tables for adults were very largely extrapolated below 5' 2" and above 5' 11", by reason of the small amount of material available for study at these extremes of height and, on that account, had been developed without regard to the build tables for youths.

This is an attempt to co-ordinate the adult tables, with the Wood-Baldwin Tables of the Metropolitan, with Drs. Frankel and Dublin's study of heights and weights of New York City children, and with certain data recently accumulated in the office of the New York Life regarding the heights and weights of youths of 10 years of age and upward. An examination of the material thus brought together, side by side with that of our adult standards, showed that it should be possible, without doing violence to any of them, to develop a graphic table which should co-ordinate the heights and weights of youths of 10 years of age and over, with those of adults, as already, in the main, so firmly established. To accomplish this, it was thought to be justifiable to assume that the age curves representing the various ages from 10 to 20, should in fact, run nearly parallel to one another, only diverging slightly in proportion to the normal increase in height and weight. A reasonably well established curve at age 10, sup-

ported by a similar curve at age 15, should serve to give a better idea of the trend of weights below 5' 2" at age 20 and above, than the extrapolations which have been used in the preparation of the older tables. This Graphic Table is the result. At the younger ages it agrees within reasonable limits with the sources referred to above, and at the older ages, between 5' 3" and 6' 0", it is in very close agreement with the old standards. Below 5' 4", and above 5' 11", the age curves of the old tables have had to be quite sharply depressed in order to bring them into harmonious relations with those of the younger ages. At 4' 9", for example, there is a difference between the old curves and the new of nearly 10 pounds—a change, which, while it is considerable, is necessary in order to bring the two sets of tables into harmonious relations.

It must be remembered, that the weights given in this table are not net weights, but are weights in ordinary clothing, without coat and vest.

Since the practice has become so general among the life companies of extending the benefits of life insurance to quite young people, we have all felt, I think, a good deal of embarrassment in passing judgment upon risks because we had no very definite means of determining what the build of the individual was. This is an attempt to bring into line with our well established tables the build of young people. I cast about for data on which to base this table and the best thing that I was able to find was a table that is in use in the company of my venerable and esteemed contemporary here, Dr. Willard. That table seemed to be the best thing that was in sight. Then, Dr. Frankel of the Metropolitan and Dr. Dublin had made a study of New York City children and had worked up the builds for two age groups with a good deal of accuracy. In addition to that, we accumulated in the New York Life quite a bit of material bearing on the build of our young applicants, and this is an at-

tempt to weld that all together and bring it into line with the standard table.

I want you to recall to mind the composition of the table that we are all using in our office. It is based upon very trustworthy data down to about 5 feet and 3 or 4 inches and up to about 5 feet 11 inches, but outside of those limits both above and below the table is untrustworthy because it rests on a very small amount of data. Now at the lower levels, at 5 feet 3 and below 5 feet 3, our table, our medical-actuarial table, was so far at variance with the table of young people that it was perfectly evident that something had to be done to bring them into line. Those of you who have made use at all of the old graphic standard table of the New York Life will remember that from 5 feet and 3 inches down the line curved quite sharply toward the left. In order to bring that table into harmony with the table which seemed to be pretty fairly established, it was necessary to depress the lower end of all of those curves and the result is this graphic table which you have before you. I believe that it will prove to be pretty fairly dependable.

Notice that it begins at 3 feet and 11 inches and that the curve which is labeled 10, that is, the age 10 line—and it applies to males and females alike. The next curve above applies to males for age 15 and females for age 18. The age 20 line for males is the age 26 line for females. Now, in the practical working of this table, let us assume that a person is 4 feet 7 inches in height. Notice the vertical black line labeled 4 feet 7 inches. The weight for a person 10 years of age, either male or female, is where the vertical line labeled 4 feet 7 inches intersects with the age line 10. Follow the 4 feet 7 line down until you come to the age 10 line and that shows you that the weight of a person, male or female, at 10 years of age and 4 feet 7 inches in height should be 75 pounds. If you follow that same 4 feet



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7 line upward, you will find that at age 15 the 4 feet 7 line indicator is intersected by the weight line 85 pounds, which means that males 15 years of age should weigh on an average 85 pounds. Of course, for any intermediate age between age 10 and age 15 you will fix upon the point that distributed that distance evenly, that is to say, a person 4 feet 7 inches in height, 12 1/2 years of age, should weigh 80 pounds for boys and for girls it would be a little lower than that.

There is one point further that I should like to touch upon, and that is this: Let us suppose a person is 5 feet 2 inches in height and is, say, a woman 26 years of age. Follow the 5 feet 2 line down to the intersection with the age 26 line and you find that such a person should actually weigh 120 pounds. Let us suppose that instead of weighing 120 pounds that person weighs 175 pounds. Follow the horizontal line 120 out toward the right until you come to the vertical line marked 175 pounds and you will see that that person is 45 per cent overweight. The diagonal percentage line 45 per cent meets just at that point, so that you can determine pretty nearly any degree of over or underweight by fixing upon the average weight and then passing horizontally to the right or left until you come to the exact weight of the individual and you find there a percentage line which will give you the degree of over or underweight.

Dr. Hobbs—I think especially at the younger ages we have been somewhat at sea in regard to computing the percentage of under or overweight. I am quite sure that this table will be of great benefit to us all.

Dr. Rogers—Dr. Eakins raises the question whether I have made the point clear how these basic weights are determined, and I shall be very glad to go over it again to help anyone who has not caught onto it. Let us take, for example, the 5 feet 3 line. A person 10 years of age and 5 feet 3 inches in height should weigh, or rather the average weight of such



## Rogers—Graphic Standard Table 49

a person is 105 pounds. The horizontal line gives you the weight, 5 feet 3, age 10, 105 pounds. Take the line 5 feet 2. A person 5 feet 2 inches in height, a man 20 years of age, weighs 120 pounds. I selected those intersections because they are more easily read. A man 5 feet 8 inches in height and 40 years of age should weigh 160 pounds. This is for normal people, not extraordinary people. Have I now made it clear? Beyond age 50 the weight, if anything, falls off a little bit, and remember, this is not the best weight. A number of years ago, Dr. Symonds, one of the most valuable members of this Association, urged upon the Association to adopt a standard table of heights and weights based upon the best weight, and in some respects I am sorry that the Association did not decide to do that. The difficulty would lie, however, in the fact that many of our offices have been doing business for many years on the old standard of heights and weights and they speak of the over or under men. Now the average weight, the best weight only at about age 38 or 40—at age 15, for example, the best weight is about 15 per cent over the average and at age 60 it is about 15 per cent under the average, and the intermediate best weights in between. This table is based upon average weights and it is based on the weights of people just as they stand before you without coat and vest.

Dr. Hobbs—Are there any questions?

Dr. Weisse—I have been more or less interested in weights, principally overweights, for a good many years, and I want to thank Dr. Rogers for his contribution in weights and percentages which he has given us. I think he has shown a great deal of ingenuity and certainly a great deal of thought.

Dr. Hobbs—I think all of us will find this table useful. The afternoon session will take place at 2:00 o'clock, after luncheon is over. Luncheon will be served immediately.

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### AFTERNOON SESSION

Dr. Hobbs—We will now proceed with the afternoon session, and I am very glad to state that we have with us today—we will give him first place on our program for this time—Dr. James Ewing. Dr. Ewing is known to a great many of you personally. He is known to you by reputation for his eminent work in pathology in connection with the Cornell University Medical School and also the Memorial Hospital here in New York City and his general interest in pathological matters. He has kindly consented to address us on the subject of cancer with as much reference as possible to the relation of cancer to life insurance. Dr. Ewing.

### THE CHANCES OF DEATH FROM CARCINOMA

#### DR. JAMES EWING

There are some general considerations which affect this question. We all know that 1 man in 8 and 1 woman in 7 at the age of 45 die from cancer; that in the United States there are about 100,000 to 125,000 deaths per year; that there is a considerable body of untouched cancer material in unrecognized cases of cancer. Cancer is second or third in the list of causes of death, heart disease being first, but when we consider the rather miscellaneous group of cases that reach the statistical tables under this caption, we can probably assume it to be of lesser importance and that cancer and tuberculosis are the two most important causes of death today. Moreover, sound observations indicate that the recorded death rate from cancer is increasing to a variable degree in different communities and to a very slight degree in nearly all, and it appears, especially from the studies of your colleague, Dr. Dublin, that when corrected for life periods, the number of persons who reach the cancer age by virtue of escaping the diseases of early life through improved hygiene are suffer-

ing more and more from cancer—so that it is a real increase from a statistical standpoint.

However, it is important in this matter to know that not all forms of cancer are increasing. The inaccessible forms are increasing, according to statistical records, rather than the external or accessible forms. Cancer of the stomach and cancer of the breast show no definite increase, and in many statistical tables, they show a decrease. The best information I can get about cancer of the uterus indicates that it is about stationary. So that a sweeping statement that cancer on the whole is increasing may be of some general importance to life insurance men, but from the strictly medical and pathological standpoint, it is of no great significance, because some types of the disease are increasing, others are stationary, and still others are probably diminishing.

I think that most statistical studies of this subject still underestimate the extent of the untouched territory in cancer deaths, that is, those cases that are not recognized. At the recent Cancer Conference at Lake Mohonk, DeVries of Amsterdam reported on 1,200 autopsies from a general city hospital where the patient came to autopsy with a diagnosis of cancer or was found to have cancer. Of these 1,200 cases, 220 or 20 per cent were unrecognized, whereas 104 that came to the autopsy with a diagnosis of cancer were proved not to be cancer. A 20 per cent increase was to be expected in that institution from unrecognized cancer cases, but this 20 per cent was reduced to 10 per cent by wrong diagnosis. I am quite sure that the average success of physicians in this country in diagnosing the disease is no better than it is in Amsterdam, and, in fact, I have an impression it is not quite so good. So we still have a considerable increase in the recorded death rate to attribute to improved diagnosis.

Cancer is a disease of habit, and as life becomes more strenuous—as I believe it is becoming—we may expect more

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of a break-down of the tissues due to the various forms of chronic irritation, and I personally see no escape from the conclusion that the stress of life, increasing as it does from decade to decade, is going to bring a larger number of cases of cancer in persons who reach the cancer age. I also think from my experience at the autopsy table and at the institution I serve, the Memorial Hospital, that cancer is more frequent in younger ages today than it was 25 or 30 years ago, the causes of which I have been wholly unable to determine. I am sure we see cancer of the breast in younger women more frequently and malignant tumors of children seem to be more abundant than they were when I was a medical student.

What factors are there which would tend to limit this increase and especially to limit the fatalities from cancer? There are several. In the first place, as you know, there has been launched a systematic and very competent campaign of educating the public, and it was on this subject especially that the conference at Lake Mohonk was gathered. I was very much impressed to see the scope of the organization of these cancer control societies in nearly all of the civilized countries represented. While America has perhaps the oldest and in some respects the most efficient organization, the other countries, especially France, have undertaken this work on a very broad and intelligent scale, and the same is true of Sweden, Germany, Denmark, Belgium, England, where the work is now vigorously pursued. The conclusion generally reached in those countries is that the people shall no longer remain ignorant of the early signs of cancer. I don't know how much we may expect from this campaign, but there are certain indications that it has already been more or less effective in bringing people to the surgeon early with a better chance of cure, but the effects are not the same in all groups of the disease. In the external or accessible cancers, where the pa-

tient himself may see the early signs, the people come earlier. For instance, in breast cancer, we see an increased number of women with early cancer of the breast and with precancerous conditions and chronic mastitis than we did ten years ago, whereas with cancer of the uterus I believe there is no difference whatever. Women don't seem to be able to recognize the signs of uterine cancer. There is every reason to understand why they should not, because, as a matter of fact, there are no early symptoms. With cancer of the skin and the inter-oral group, people come in earlier and are getting better results, but I think it will be a long while before the campaign of education, however vigorously pursued it may be, will show itself in the statistics which you meet in life insurance work or which the United States Census Bureau is able to gather in its tables, and the reason, I think, is indicated by an incident which was reported to me by one of our English visitors at Mohonk. He was remonstrating with a colleague and told him he ought to take particular care and have periodic examinations for cancer. He said, "Why, my dear fellow, one man in eight at your age dies of cancer." The other replied, "Oh, one man in eight? Well, that's pretty good odds; I guess I'll take them." It is a fact that you can inform people about all these early signs of cancer, but it is quite a different matter to get them active. However, there have been definite improvements reported from different parts of the country as a result of the campaign of education and I believe it will be an increasingly important force in diminishing the mortality. I may here perhaps appropriately question if it is not possible for insurance men and insurance companies to lend themselves to this campaign. Your efforts certainly would not be lost.

There is another phase of the educational campaign in which I am personally interested and which is not much discussed and that is the prevention of cancer. Certain cancers

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are distinctly preventable and fortunately they are of the major type. Others are not preventable in the present state of our knowledge. These are mostly the inaccessible and uncontrollable forms of cancer. We know that if bad teeth, tobacco and syphilis were eliminated inter-oral cancer would largely disappear. All these factors are very tangible, easily understood by men of ordinary education and controllable. Inter-oral cancer is, therefore, a distinctly preventable type.

In recent months, observations have been made in regard to mammary cancer, which suggests that it may be possible to establish a proper hygiene of the breast and reduce the incidence of mammary cancer. Dr. Bagg at the Memorial Hospital has succeeded in producing cancer of the breast in mice in a large proportion of cases. By breeding animals very rapidly, withdrawing the young at birth, and allowing the milk to stagnate in the breasts, in a series of animals which had a very low incidence of cancer, he succeeded in producing cancer of the breast in 85 per cent of the experimental animals. In some instances he ligated the ducts on one side and cancer developed on that side and not on the nursed side. When we apply this principle to human mammary cancer, we find that a very small proportion of women with mammary cancer give anything approaching a normal lactation history, and we are now pursuing, perhaps too confidently, the theory that the main, effective, exciting cause of mammary cancer is stagnation. If so, then we must teach women that the breast doesn't always take care of itself but under many circumstances must be taken care of by themselves and their physicians, and the basis is laid for the hygiene of the breast to lessen the incidence of mammary cancer.

One might go down the list, but it would take a long discussion to show how many tangible exciting factors there are in the different forms of cancer. Most of the major forms of cancer are due to chronic irritation of one form or an-

other. All that group is more or less preventable, and I anticipate considerably more response from the educated man and woman in the effort to prevent the disease than in the attempt to have it cured when it actually exists. Unless I greatly mistake human psychology, that will appeal to people much more than facing a serious operation for established cancer. So from these points of view, the educational campaign on the early signs of cancer and on the prevention of cancer may, in the course of the next twenty years, have a perceptible effect on cancer mortality.

Now as to the origin of cancer. I don't know of a single experienced pathologist who regards the parasitic theory seriously, and until there is some new chapter in the biology of the cell, I don't think that this theory is acceptable. I think that there would be very few votes against that very positive assertion. If cancer is due to an unknown, microscopic parasite, then what is the use of talking about prevention? It is unfortunate that so many men in high places begin their annual orations with the old announcement that the cause of cancer still remains undiscovered. The ultimate cause of cell growth is undiscovered and probably always will be. It would be far better, it seems to me, that we emphasize the fact that we do know the effective, exciting factors of cancer.

Is cancer hereditary? Here we have the lines drawn rather sharply. Some schools, especially the experimentalists, uphold the theory of hereditary transmission and others deny that heredity is a practical factor. We, who take the latter view, lean rather heavily upon the attitude of the insurance people, who, I understand, pay no consideration whatever to a history of cancer in the family. I have no doubt that that is a sound business principle, but I think it is a somewhat sophistical argument regarding the importance of heredity, because it may very well be that in the large numbers of



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people whom you handle, it may not be worth while to discard people who have a bad family history. It seems to me that there is a distinct influence of heredity in certain forms of cancer and that it would perhaps be worth while from a business standpoint to make a special study of the hereditary influence in different forms of the disease. I do not know whether the literature of life insurance has ever provided any specific study of this sort. You discuss the influence of heredity on cancer as a whole. I do not know that there has been any particular study of heredity in specific forms of cancer. I think it would be highly important to have such data.

There is a matter of very considerable importance, from a life insurance standpoint, in the significance of precancerous lesions. Cancer very seldom develops in previously normal tissue, but nearly always in tissues altered by chronic inflammation or injury of some sort, and we have an extremely long list of lesions which we call precancerous.

The different kinds of precancerous lesions make a list too long for me to attempt to describe them in detail. I will simply point out some of the more important and give you my personal opinion as to their significance from a life insurance standpoint. One of them is leukoplakia, white spot disease, due in the majority of cases to the abuse of tobacco, aided by bad teeth and occurring in the opinions of many upon a basis of syphilis. I have watched many cases of this disease for a good many years and I confess that if they are carefully watched, they may run along year after year with recurring attacks of new white spots without developing cancer. On the other hand, unless they are watched and great care taken about the use of tobacco and especially the elimination of bad teeth, so many of these cases have developed cancer that I regard the disease as very dangerous, especially if the spots are deep and hard and tending



to horny thickening and hardening, especially also if there are bad teeth, and particularly if the patients have a harness of metallic alloys supporting teeth. Then they are bad risks. I have watched certain cases that developed cancer right under my eye and to such an extent that the disease became serious or inoperable within a few weeks. The fine small spots which appear here and there in comparatively young men with good teeth may have an entirely different significance.

Brown moles of the skin are also very disturbing. We see many of those which have been operated upon and have recurred. Yet dermatologists say that they have removed hundreds of these brown moles and they do not return. When a mole is in an exposed position, when it is black, very much pigmented, when it shows any signs of increase, when it is warty, and especially if there is any change in its character with prominence, erosion or secretion, that mole should be removed. It may already be malignant. Such a case is a bad life insurance risk as long as he has that mole. Whereas the small, flat and slightly pigmented moles are not so bad.

*Chronic mastitis.* The majority of cases of breast cancer develop on chronic mastitis. However, it would be quite impossible to assume that a young woman who has lumps in her breast should be rejected for life insurance. At the same time, if lumps begin early and if the patient is of a florid type and one that is not likely to pay very close attention to the condition of her breasts, so many of these cases develop cancer that one should be rather cautious.

Uterine lacerations after childbirth, unhealed, are nearly constant predecessors of uterine cancer. If a woman applies for life insurance with a history of childbirth, with signs of leucorrhoea, and no statement as to the condition of her cervix, I think you are taking an unnecessary risk if you do not ask for a vaginal examination and demonstration that there are no unhealed lacerations of the cervix. It might be

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impossible to demand such an examination, but from a medical standpoint it is worth while.

There is a great deal said about ulcer of the stomach developing into cancer of the stomach. Many believe that the ulcer stomach is no more liable to develop cancer than normal stomach, but others take quite a different view of this situation and you will have to decide for yourself.

This is a phase of neurofilramatoris marked by pigmentation of the skin, small tumors of the skin, or small tumors beneath the skin.

It has been estimated that eight per cent of such subjects die of a malignant form of sarcoma, which is very resistant to all sorts of treatment and we see case after case where a surgeon has carelessly removed a small apparently harmless tumor with immediate recurrence and eventual death of the patient, as a rule after a good many years of suffering. When a patient has one or more subcutaneous tumors of this type he is a relatively poor life insurance risk, although if he does develop sarcoma he is likely to live a long while.

What are the chances of death from established cancer? There are a number of major forms of cancer which give 100 per cent mortality. In some of these cases we prolong life quite a little through radiation properly applied, but not in many.

What are the chances of a woman with mammary cancer recovering from the disease? On this point, you will find the widest variation in statistical reports, from 75 per cent of permanently cured patients with early cancer of the breast down to a recent report by Moschowitz, of the Mt. Sinai Hospital, who followed for 11 years a considerable series, over 100 mammary cancers, and at the end of 11 years not one of them was alive. I find it very difficult to form any positive opinion regarding the prognosis of mammary cancer. My own feeling is that when a woman has had the disease,

she has about one chance in thirty of living five years, and practically no chance of dying without the disease, although her life may be so prolonged that she may be a fair life insurance risk. Much, but not all, depends on the treatment she receives. With early localized breast cancer, the statistics are much better, but probably do not run over 10 or 15 per cent. I have no doubt this statement would be rejected at once by the surgeons who are publishing these very favorable reports from their clinics and who claim 40 or 50 per cent of permanent cancer, but from my observations, I believe that the mortality is high even with early localized mammary cancer. I understand that the different companies apply different rules in regard to supposed cures of mammary cancer, some rejecting them altogether and some waiting five years and giving them a high rate, and some waiting ten years and also giving them a high rate.

*Uterine cancer.* The advent of radiation has changed the aspects in uterine cancer considerably and gives us about the same success with radiation as was obtained by surgery without mortality. In addition it lengthens the life of those that recur and of those that are quite inoperable. The duration of life in uterine cancer is much greater than it was before the advent of radiation.

*Stomach and rectum.* Stomach cancer is practically a fatal disease. I should say from a life insurance standpoint there are no grounds for insuring a man's life who has had an operation for gastric cancer. Rectal cancer is also a very fatal disease and while there has been some improvement in recent years, it still remains a grave problem.

In the case of lip and tongue, inter-oral cancer, the prognosis is considerably better than it was. We treat all lip and tongue cancers at our clinic with radium and that rule is followed by other clinics in Stockholm, Paris, elsewhere. The results are good and recurrences are less frequent. The

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number of cases saved permanently and those that have their lives distinctly prolonged is considerably greater than before the advent of radiation. Nevertheless, this is such a treacherous disease, that any one of them is apt to recur. Or after one cancer has been cured another will develop one. The mouth once condemned by cancer is very prone to develop another, so I am not optimistic at all about the wisdom of considering cured cases of lip or inter-oral cancer as good life insurance risks.

Radiation is bringing about the prolongation of the lives of many patients and enabling them to live a normal life for a period of years. Inter-current diseases then come in and they are signed up for the inter-current disease and not for the cancer. I have several times performed autopsies on persons who had cancer but who died of inter-current disease, the cancer being temporarily retarded or suppressed, and as that period of suppression increases, the mortality assigned to cancer will be lower.

One more point in closing: It seems to me that the statistics about cancer in general are very limited in value. All our knowledge and our views would be very greatly clarified, if we regard a cancer as a great group of special diseases connected by the fact that they are all marked by malignant overgrowth of tissue cells but caused by a great variety of different forms of irritation and many other contributing factors running different courses and requiring different forms of treatment to which the response is very variable, and with histories which are so different that we must regard them as separate clinical and pathological entities. Until we take this point of view, we shall be working in the dark and not reaching conclusions which are really important. The observations on cancer as a whole may be very interesting from a broad statistical standpoint, but from the personal standpoint of the individual whom you may be undertaking to insure or whom

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doctors in practice are undertaking to treat, observations on these diseases will only be valuable when they refer to the different forms of cancer and not to cancer as a whole. Is it not possible for the insurance companies, statisticians and medical departments, to attempt much more detailed study and observation upon the specific forms of cancer and avoid as far as possible grouping the whole disease as one?

Dr. Hobbs—You have heard a very interesting address by Dr. Ewing. I do not see that there is much for us to hope for, as far as insurance is concerned, in insuring lives with a history of this disease. I do think that his remarks concerning pre-cancerous conditions should receive serious thought from us, when we are selecting our risks.

I am quite sure that Dr. Ewing will be glad to answer any questions, but before that, we will have the discussion. I will call upon Dr. Knight to say a few words to us.

Dr. Knight—Mr. President and Gentlemen: I certainly am very much embarrassed in attempting to make any discussion of this admirable address of Dr. Ewing, without having known in advance what he was to say. He, as one of the leading pathologists of the world, has told us about the research work that is being done, and about the ways of preventing cancer and of curing it in its early stages. That Cancer Committee, of which he is a member, is doing just as fine a piece of work as probably can be done anywhere, and it certainly has a promising future in front of it. It is accomplishing a lot, is going to accomplish a great deal more and the question comes up as to what we can do about it. I happen to know that just at the moment that Cancer Committee is undertaking to raise a million dollars to carry on its work of research and publicity and that it is very active in that endeavor.

They have raised perhaps \$350,000 and a great many members of the laity are saying: "These rich life insurance com-

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panies should give \$100,000 or \$200,000 to that fund." I was even asked the other day whether it wouldn't be a good thing to have representatives of the committee go before the companies and ask for donations. I told them that they wouldn't and couldn't get a favorable response to such a request, for as you and I all know perfectly well our company executives do not feel that they can take money which they hold as trustees and give it away to other organizations, even though it be for the best of purposes. There has been very little attempt by any companies to do anything of the kind. I know that in our own company some years ago, when Dr. Gaylord was carrying on his cancer research work in Buffalo, our chief counsel wanted very much that the company should subscribe to the promising experiments, and I think that the sum of \$5,000 was given. I haven't heard of anything else being done along that line except that the John Hancock gave something to the Crocker Fund for cancer research two or three years ago. The money that the companies hold belongs to the policyholders and I don't believe for one moment that we could get our executives to say that it is legal for them to give that money away.

I do think, though, that there is an awful lot of helpful work that we might do if we plan it rightly, and I think that it ought to be work that is mapped out and planned and put before us by this very Cancer Committee. They can prepare the materials for it very much better than we can. I don't think we ought to carry on that work out of our own initiative.

Dr. Ewing asked whether any work had been done by any of us to determine whether cancer is hereditary and on looking back through the transactions of our own society I find that absolutely nothing is shown in the reports. The only report at all is the paper that Mr. Hunter presented to the Life Insurance Presidents' Association in 1916. That

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was ten years ago. There he had 488 cases, and from the study of those 488 cases he said that it didn't seem as if cancer is contagious or is hereditary. Dr. Ewing has indicated that it would be worth while to find out whether it is hereditary. I think his opinion is that it would be a worth while piece of work and if that is so, that this Association might very well record itself as being in favor of making such a study. It might well be done by the Medico-Actuarial Committee. I think, therefore, that we can be of substantial help to the Cancer Committee if they will get together such material as they want to put before the public about the ways of preventing and curing cancer and if they will then give all of our member companies, the opportunity of printing and distributing these messages widely among the policyholders. In this way each company can thus pay for its own printing and for its own distributions and I think that all of our companies may well be in favor of such an undertaking, and that it would be well worth while in its results, and to my mind it is the only way in which we can or ought to ask our companies to assist in this work.

Dr. Hobbs—Dr. Toulmin.

Dr. Toulmin—Mr. President and Gentlemen: I should like to join with Dr. Knight in saying to Dr. Ewing that his address was not only interesting, but—and I am sure you all agree with me—was extremely instructive and very practical in its application to our work.

I had the good fortune yesterday of attending a luncheon given by a prominent lawyer of New York, Mr. Aldrich, in connection with the work being done by the American Society for the Control of Cancer. Addresses were made by Doctor Howard C. Taylor and Doctor Francis C. Wood. It was pointed out that the cancer problem, broadly speaking, was being attacked in three ways: First, in the form of research work; second, in the form of treatment; and third, in the



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form of educating the public. We were made to understand very distinctly that educating the public included educating the physicians of the country. I believe that it is in helping this society to extend their ideas to the public at large that every life insurance company can be of really immense value.

Within the last five years, we distributed among our policyholders certain leaflets calling the attention of the recipient to some of the early signs of cancer. The necessity of having such information brought to the attention of everybody possible is very, very strongly presented in statistics collected by Hoffman<sup>(\*)</sup>. He states, for example, that of 62 deaths of males of cancer of the tongue only 12 had been operated upon at the time of death; of 13 cases of cancer of the lips, 6 had been operated upon; and of 49 cases of cancer of the neck, 21 died without being operated upon.

Doctor Wood told us of a campaign that was put on in Detroit by their society, one result of which was that it brought to their clinics over 200 cases of cancer in which the individual had not consulted a doctor. Do not such experiences positively indicate the importance of educating the people as to the early signs of cancer, and the importance of early seeking proper medical advice?

The three outstanding ways in which we can assist in this problem are: First, contributing money ourselves, or having our friends contribute to the fund of \$1,000,000 which the Society for the Control of Cancer is raising; second, getting contributions from the companies we represent; third, by circularizing leaflets or letters of the society and by this means place valuable information in the homes of millions

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(\*)—"San Francisco Cancer Survey—Second Preliminary Report (Fourth Quarterly Report)." July 1, 1925—by Frederick L. Hoffman, LL. D.



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of our population and in the offices of thousands of our leading physicians.

I cannot urge you too strongly nor too earnestly to aid in every way possible to bring about a favorable solution of this very important problem.

Dr. Hobbs—I would like to ask Dr. Ewing what he considers the chances of epithelioma of the lip eradicated either by radiation or surgery, whether five years would be enough to consider that person practically cured.

Dr. Ewing—Yes and no. A great majority of cases, about 80 per cent, of fairly early carcinoma of the lip are controlled by either method, radiation or surgical, but there are always those unexpected recurrences. Much depends upon the care with which the patient will permit himself to be watched. In our work at the Memorial Hospital, the main difficulty in treating lip cancer with radium, which we employ in preference to surgery, is to get the patient to realize that the disease may return. They will not do it until it is in a worse condition than it was before. The prognosis of well handled early lip cancers after five years ought to be good.

Dr. Toulmin—May I ask your opinion of a case where the applicant had used tobacco to excess, had bad teeth and a history of leucoplakia, not deep-seated but rather superficial. The teeth have been corrected and tobacco is used in moderation. Do you believe that after two years' absence of the leucoplakia we might safely accept such a risk?

Dr. Ewing—Yes, under the conditions you describe, but if that man is a bad abuser of tobacco and his teeth are bad and he has colloid plates in abundance, it is almost certain to return and he will die unless you watch him.

Dr. Wehner—I would like to ask Dr. Ewing his opinion of cancer of the salivary gland.

Dr. Ewing—It is regarded as a rather mild, slow, localized disease, but our experience indicates that it is pretty fatal. It

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occurs locally and is very resistant to operation and it is resistant to radium. It recurs in increasing malignancy and we regard it as a dangerous form of cancer.

Dr. Frost—I would like to ask Dr. Ewing his prognosis of papilloma of the bladder which has been removed apparently successfully without signs of malignancy.

Dr. Ewing—Statistics show the papilloma of the bladder is fatal in 33 per cent of the cases. They usually recur in the same form, and the transformation from a benign to a malignant form is so rare that the authorities quarrel about its existence, so that the chances of recurrences are good and the chances of death from the disease are poor.

Dr. Old—How about gallstones? Does cancer follow gallstones?

Dr. Ewing—Nearly all gall bladder cancers occur in cases of gallstones and the removal of the gallstones prevents the cancer in nearly all cases.

Dr. Old—Have you run across any cases of cancer that follow operation for gallstones, say, after three or four years?

Dr. Ewing—They have their cancer of the gall bladder and their gallstones at the same time, but I believe that the removal of the stones is an important method of preventing gall bladder cancer. However, cancer of the gall bladder will arise after removal of the stones, but I don't know how often as I don't have any statistics.

Dr. Honsberger—We have a great many cases of warty growths on the lip which have been removed and are supposed to be innocent. Do those usually become malignant? Removed possibly six months ago or three months ago and are said to be innocent.

Dr. Ewing—All warty growths on the lip are suspicious. There is a distinct variety which remain benign for a considerable period but eventually will become malignant.

Dr. Hobbs—Are there any further questions to be asked

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of Dr. Ewing? I think that we have enjoyed Dr. Ewing's talk to us and I also think that we owe him a vote of thanks for the subject that he has so ably covered and for the information that we have been able to get out of his remarks.

As I said this morning, Dr. Rockwell has a paper here, "Does the Insurance Record Affect Selection?" The paper is one which I think will be of value to us all and I hope the discussion of that paper will be valuable. After the paper of Dr. Rockwell, we will proceed to the paper of Dr. Muhlberg. Dr. Rockwell.

### DOES THE INSURANCE RECORD AFFECT SELECTION?

BY THOMAS H. ROCKWELL, M. D.

*Medical Director, Equitable Life Assurance Society*

In the consideration of applications coming to us day after day I think we sometimes wonder whether or not we profit by information that may be on file concerning the applicant, or whether from his statements and our examiner's report, we may not have sufficient knowledge to permit us to act intelligently and satisfactorily.

When we consider the favorable experience that group and non-medical selection has given for those insured under comparatively small amounts, computing the medical, inspection and other underwriting expenses, the premium loading for these expenses might reasonably be expected to take care of a certain amount of increased mortality, due largely to individual antiselection. On the other hand if it should become universally known that medical examinations were to be done away with under certain amounts, a part of the insuring public would soon become wise, and the premium loadings referred to might not be sufficient.

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As Insurance is now written I feel all reasonable safeguards should continue and it is well for us to enquire from time to time into the relative values of the tools we are accustomed to use. Hence I have made a study of consecutive applications as they came to us day by day from June 17th to July 10th, 1925. 13,456 *approved* medical examinations were made. Of these 1,056 or 8% had records of previous impairments. Of the 1,056 cases our *action* was influenced in 495 (47%), not influenced in 561 (53%). By *action* is meant that papers were delayed going through in order to permit further inquiry. This means, re-examination of heart, lungs, urine, detailed history of past illness, further inspection or whatever criticism not covered by the examiner or inspector. In 561 (53%) we felt we need not delay our action because our examiner or inspector had covered the points of criticism. When it came to the final rating and the ultimate disposition of the application, we found such rating was affected in 169 (16%) and *not* affected in 887 (84%); that is, the information we had in 169 cases (16%) was sufficient to place the policy in the substandard class.

### APPROVED EXAMINATIONS REVIEWED

With impairment record	1,056— (8%)
Without impairment record	12,400— (92%)
<b>Total</b>	<b>13,456—(100%)</b>

### APPROVED EXAMINATIONS WITH IMPAIRMENT RECORD

Action affected	495— (47%)
Action not affected	561— (53%)
<b>Total</b>	<b>1,056—(100%)</b>
Rating affected	169— (16%)
Rating not affected	887— (84%)
<b>Total</b>	<b>1,056—(100%)</b>
Issued standard	616— (58%)
Issued substandard	440— (42%)
<b>Total</b>	<b>1,056—(100%)</b>

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### DECLINED EXAMINATIONS REVIEWED

With impairment record	278— (29%)
Without impairment record	672— (71%)
Total	950—(100%)

### DECLINED EXAMINATIONS WITH IMPAIRMENT RECORD

Influenced final action	63— (23%)
Not influenced final action	215— (77%)
Total	278—(100%)

The declined cases (278), together with the accepted standard or substandard (1,056) amount to 1,334 in which a previous impairment was recorded.

Of the 440 substandard cases our action was not influenced in	43— (10%)
Simple impairments influenced our action	82— (19%)
More than one impairment influenced our action	315— (71%)
	440—(100%)

Final* rating—	
Not influenced	115— (26%)
Single impairment influenced	69— (16%)
More than one impairment	256— (58%)
	440—(100%)

Of the cases affected in final rating (325) I formed arbitrary classes from 100 to 800.

100—General	19— (6%)
200—Nervous	12— (4%)
300—Circulatory	89— (27%)
400—Respiratory	21— (7%)
500—Digestive	21— (7%)
600—Genito-Urinary	124— (37%)
800—Miscellaneous	39— (12%)
	325—(100%)

In each of these classes are included what appeared to be of most importance and relative frequency. In Class one: A history of syphilis, rheumatism, or malignant growth. Class two: Epilepsy, insanity, attempted suicide, nervous disease. Class three: All circulatory diseases and blood pressure. Class four: Tuberculosis, lung diseases or pharyngeal

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trouble. Class five: All digestive troubles. Class six: Albumin, casts, sugar, kidney colic and all other kidney or genito-urinary diseases. Class eight: Criticism of habits, character, moral hazard, insurable interest, appearance older or unhealthy.

In the 1,334 cases, 1,817 impairments were listed and distributed as follows:

One or More Impairments	
Accepted 1,056	Declined 278
1      111	1      31
2      27	2      4
3      413	3      142
4      153	4      27
5      125	5      21
6      420	6      95
8      176	8      72
<hr/> 1,425	<hr/> 392

In the cases rated as substandard our *action* was influenced by the 600 class more than any other. This, of course, required often repeated urinary examinations and sometimes certificates from operating surgeons or attending physicians.

The 300 class was next in volume in which we requested additional examinations of the circulatory system.

The 200 class, though small in number is very important, necessitating often a re-examination or more especially, a letter from the attending physician or hospital records. To me, the 800 class seems the most important of all. This is usually the interpretation of confidential reports and frequently they are voluminous. In my own company this class represents our worst mortality and those we have taken at substandard rates have given us the highest rate of any class.

Where an individual is rated up for a physical impairment it is comparatively easy for the agent to sell the policy. But when it is for habits, moral hazard or environment there is often a resentment on the applicant's part. The agent is not

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in full possession of the reasons for our action and many times can only surmise the real reason. So his "talking point" is curtailed and if the policy is placed the chances are that our rating in that particular case was too low, and this results in antiselection against the company and its accompanying high mortality.

From an underwriting angle, these cases are the most difficult to judge and to assess a correct rating. Hence, a comparison of the reports obtained from different sources is all important and companies should do all in their power to protect each other.

Of the 63 declined cases in which we were influenced almost solely by the impairment record, one (2%) was in class one, three (5%) class two, eleven (19%) class three, two (3%) class four, four (6%) in class five, fifteen or (23%) in class six, and as we might expect, twenty-seven or (42%) in class eight.

When an applicant gives a considerable list of policies, previously received in this or other companies, showing repeated examinations, and when there is no impairment record on file we are very likely to give some credit for this in our consideration of the case.

Then, again, if we find a long record of some impairment, especially the heart, showing no increase in the lesion after many years we are apt to look more favorably on our prognosis.

Approximately, I can state that this year, because of the impairment record, the company that I represent will get a higher premium on close to ten million dollars' worth of new business, and will save, on this year's business alone more than \$100,000 on mortality losses.

From the above brief study and analysis, I feel the possession of previous information concerning an applicant is all important, and that members of the Association of Life In-

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surance Medical Directors should continue to help each other and the companies they represent in every legitimate way.

Dr. Hobbs—Dr. Van Kleeck.

Dr. Van Kleeck—Mr. President and Gentlemen: One is loath to discuss a paper such as Dr. Rockwell has offered us because the facts so ably presented lead inevitably to the conclusion of the final paragraph. The thought does come, however, that the insurance record is valuable not only because it helps to mark the impaired risk, but also in that it enables us to save certain business. We can often accept either standard or rated those individuals with present impairments which, from the record, are either stationary or of dwindling importance in their adverse relation to longevity. Without the record, postponement or rejection must have been their fate.

Similarly the record is sometimes analogous to a consultation of physicians over a baffling diagnosis of an obscure lesion for it brings together the opinions of different examiners expressed at different times and helps to a final judgment.

While not properly belonging to a discussion of Dr. Rockwell's data, a few items secured by review of one thousand consecutively filed applications to our company may be of interest.

### 1000 Applications in Order as Filed

		Approved Standard	Approved Substandard	Postponed Or Rejected
Action at first reading	81%	726	19	62
Action after investigation	19%	29	9	135
Delayed 193	(1) Because of record			174 or 90%
	(2) Because of questionable items in application			19 or 10%

Of 193 delayed action cases but 19 or one-tenth were held up because of some statements in the application. (Unfortunately I overlooked the chance to ascertain the number in which the record cleared such a questionable feature and avoided delay.) The other nine-tenths, 17% plus of the en-



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tire one thousand, were delayed because of facts otherwise recorded.

Of the 1,000 cases reviewed 530 had previously filed applications in our company, 282 in the Life Department, 248 in the Accident and Health Department. 533 had previously applied to other (life, accident or health) companies. These groups show considerable overlapping, of course.

Out of the 1,000 cases there were 112, over 11%, on which we entered a record of an active impairment.

Truly we could not carry on so well without the insurance record.

Dr. Hobbs—Dr. Pollard.

Dr. Pollard—Mr. President and Gentlemen: Are we benefited by having the previous record to aid us in arriving at a conclusion regarding our action? Would we be willing to give up our Bureau or take our cases without any reference to previous records other than those obtained in the declarations to our examiners and in our previous examinations?

We all realize that occasionally an uncertain examiner or one who has not the courage of his convictions will re-examine an applicant and find something at the time of his second examination which he did not find originally; simply because we tell him what to look for, but take the case of the examiner who is sure of himself; do his present negative findings always indicate that an applicant is cured of his previously reported impairment or is it in an intermittent stage? If he had some heart impairment which is now apparently cured, is it not possible that it may recur and is he not possibly an understandard risk? Is it not possible that he has fixed himself up for our examination and does not the previous record give us a line which we ought to take cognizance of even though our examination shows nothing abnormal?

We know that impairments are frequently reported incor-

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rectly by the examiner and by the companies to the Bureau, but, even so, they raise the red flag of danger and make us more careful in our selection.

Take it from another angle. Does not the previous record of such impairments as heart murmurs, albumin and sugar in the urine, especially if reported more than once or twice, indicate an understandard individual and one liable to a recurrence, even though these impairments are not found at the time of our examination; especially so if the previous record is of recent occurrence? Of course, this applies only to such previous records as are thus indicative of a chronic condition. We, in our company, consider such previous records, when the identity is clearly established and there is no doubt regarding the presence of the indicated impairment in the applicant previously, as we feel that a group of such cases would show a higher mortality.

It seems to me that no one would dispute the statement that a person who breaks down or shows some weakness under strain is not as good a risk as one who does not. How, then, must we regard an individual who has had some impairment found a number of times in the past, even though at the present examination he may seem normal and his impairments not have been found at every examination?

It seems to me that our greatest difficulty in using the previous record is how much value to give same. Take, for instance, the heart murmurs. When an aortic regurgitant murmur has been reported and our report shows a mitral regurgitation are we bound to think the applicant has had an aortic regurgitation or think the examiner has made an error in diagnosis and, if so, which examiner?

For example, just recently a case came to me in which one of our examiners had reported both aortic regurgitation and aortic stenosis, with moderate hypertrophy. This was in 1921, and later other companies had reported heart murmurs,

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but no aortic murmurs. Our present examination is clean (two examinations made at our request show no cardiac impairment) so we must decide whether the previous records extending over a period of four years indicate any impairment or a functional condition which has cleared up and that the applicant is now in normal condition. We feel that the fact impairments were found over a period of years, while they may not have been accurately diagnosed, indicate that the applicant under strain is apt to develop some fixed chronic condition and become a substandard risk, and this should be taken care of by an increase in the rating.

Impairments which have been reported only once and cleared up, or twice at close intervals and not found at this time we do not consider in our rating when our report is a favorable one. Of course, the records which give us all the most trouble are the urinary and moral hazard, and in consideration of the moral hazard it is largely a matter of personal opinion and changes from year to year. We can all remember that it used to be considered suspicious of speculation if an applicant made a child the beneficiary when the child was of a wage earning age. And how much reliance can we place upon a man's word regarding his drinking habits? Rarely do we find a man who will admit he drinks to excess and we must, of necessity, take into consideration the previous record, as well as our mercantile reports. Much less often, but still frequently, an applicant will deny that he has known anything about albumin or sugar having been found previously, or that he has been on a diet or under treatment, so that we must take cognizance of the previous record that we may place him in the class in which he belongs if we give him any insurance.

As the limited time at our disposal, following receipt of Dr. Rockwell's paper, did not permit us to make an analysis of such a large number of approved cases as he did, and we

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could, therefore, make no comparison of our results along these lines; and a review of a small number would not allow us to make a fair statement, we did not attempt it.

We, however, did review some of our experiences in the albumin, sugar and heart cases, which might be of interest to mention.

For instance, during the period 1897-1922, our experience with the cases in which heart murmurs had been previously reported and not present at time of action shows that of 5,078 so reported there were 111 deaths with a mortality of 94.

Among the albuminuria cases, over a four-year period 1921-1924, those cases which had a previous record of albumin which was cleared up at the time of our examination, but which were rated at our Special A rating because of the previous record or some other feature of the examination, there were 434 cases with 9 deaths, a mortality of 188, and of these deaths 2 were from chronic nephritis and 2 were pulmonary tuberculosis, while the cases during the same period showing albumin at the time of examination and rated the same were 1,560 in number with 11 deaths, a mortality of 110.

We also made an analysis of our sugar cases which were cleared up between 1912-1922, and found there were 2,056 cases with 37 deaths, a mortality of 87, and of these deaths 3 were from diabetes mellitus and 2 cancer of the liver.

Dr. Hobbs—Vice President McCall has arrived and wants to say a few words. I know we all will be glad to hear him.

Mr. McCall—I cannot quite agree with Dr. Hobbs that I want to say a few words to you, for whenever I am called upon to make a speech, I feel very much like the Irishman who consulted our Dr. Rogers. Dr. Rogers knew the kind of life the man had been leading—so, after examining him and finding a great many serious impairments, he said to him, "If you go on leading the kind of life you are living, I will

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give you about three years to live.” The Irishman said, “Is that so. Well, do you know, Doctor, I don’t care much about *when* I am going to die, but I would give anything to know *where* I was going to die.” Dr. Rogers asked: “Why in the world do you want to know where you are going to die?” “Well,” the other replied, “if I knew where that was, I would never go near the damn place.”

Notwithstanding the gloomy outlook that President Kingsley took in his address to you some four years ago when he nearly had your positions vacant on the very day he was talking, it is a fact that your association has gone forward and onward, and today there isn’t any association connected with life insurance that has a more responsible part in the progress of our business. In my twenty-seven years of connection with the New York Life, it has been my privilege and my good fortune to have been closely associated with the work of our Medical Directors and with the work of our Medical Department, and in that connection I have watched with great pride and pleasure the achievements of Dr. Rogers and Mr. Hunter, and, in later years, of Dr. Hobbs in connection with your association. It is a source of much gratification to remember that in all this work these men have had the backing and whole-hearted co-operation of all the officers of our company.

I am sorry that we cannot meet today under the roof of our new building. I trust, however, when our new office home is completed that we may have the pleasure of having you as our guests. In conclusion, if, during your visit here, there is anything that any of you may want in the way of service at 346 Broadway, it is yours for the asking.

Dr. Hobbs—We have just heard Dr. Rockwell’s paper and the discussions of Dr. Van Kleeck and Dr. Pollard and now after the kindly words of welcome from Vice President Mc-

Call, we will go on with the further discussion. Are there any more remarks to be made? Dr. Eakins.

Dr. Eakins—Mr. President and Gentlemen: Dr. Rockwell may be interested in knowing that the company I represent made, beginning in January, 1925, and carried along at intervals to cover six full months, an investigation of its action on insurance record risks. It was done, not as exhaustively as his, and for another purpose. One set of our results is so closely parallel that I thought you might be interested. Rearranged so that they should be figured similarly, I find that 9.9% of all of Rockwell's cases had an impairment record. Of ours 11.4% were so noted. His company issued standard 46.18%, mine 49.61%; his, substandard, 32.98%; mine, 32.10%; his declined, 20.84% and mine, 18.29%. What these figures prove may be something, or nothing. They are so alike though that at least they point to over 45% of cases with an insurance record on which both Rockwell and I issued insurance with no restrictions whatever. Rockwell's conclusion, as expressed in his last paragraph, is very much to the point; but, assuming that we know how to value a risk, does it not seem that possibly unimportant impairments are too frequently used in the insurance record?

Dr. Hobbs—Dr. Fisher.

Dr. Fisher—I desire to present some data on impairments.

#### EXAMINATION BY PHYSICIAN OF APPLICANT WHO WAS APPOINTED EXAMINER OF COMPANY

We have a record of 38,550 cases, issues 1885-1908, carried to anniversary in 1915, and the mortality was 12 points better than the general average mortality of the company.

#### CANCER AND TUBERCULOSIS IN FAMILY HISTORY

We have issued 1,199 policies during the years 1885-1908,

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mortality carried to anniversary of 1915; also 874 cases, issues of 1906-1915, carried to 1920, which gave a mortality considerably below the general average mortality of the company for each period covered.

### APPENDICITIS WITHOUT OPERATION, LAST ATTACK WITHIN TEN YEARS

Number of cases issued, 1,295, during the years 1885-1908, carried to 1909, and 2,103 cases, issues of 1906-1915, carried to 1920; mortality in both groups was a few points below the general average of the company.

### REMOVAL OF APPENDIX

Issues 1885-1908, carried to 1915, 2,636 cases, and 9,285 cases from 1906-1915, carried to 1920, with the mortality slightly below the general average of the company.

### TYPHOID FEVER, WITHIN TEN YEARS

16,791 cases, issues of 1885-1908, carried to 1915, and 14,446 cases, issues of 1906-1915, carried to 1920. In both periods the mortality was several points below the average mortality of the company.

### EPILEPSY, ONE PARENT DEAD OF (OR LIVING)

505 cases, issues of 1885-1908, carried to 1915, mortality only about two-thirds of the mortality of the company.

### HEMORRHOIDS, WELL-MARKED CASES ONLY

1,199 cases, issues of 1885-1908, carried to 1915, mortality one point over the general average mortality of the company.

### BOTH PARENTS DIED AT AGE FIFTY-FIVE OR UNDER

5,509 cases, issues 1901-1908, carried to 1915, and 8,363 cases, issues 1906-1915, carried to 1920. In both periods the mortality was within the limits of the general average of the company.

## ALCOHOLIC PARENT, APPLICANT TOTAL ABSTAINER

733 cases, issues 1885-1908, carried to 1915, and 353 cases, issues of 1906-1915, carried to 1920. Early issues, very favorable mortality; later issues, about average mortality.

## ALCOHOLIC PARENT, APPLICANT MODERATE USER

450 cases, issues of 1885-1908, carried to 1915; mortality somewhat higher than the above, but below the general average mortality of the company.

## SUPPURATIVE MIDDLE EAR DISEASE

1,148 cases, issues of 1906-1915, carried to 1920; mortality 2 points better than the general average of the company.

## APOPLEXY IN FAMILY RECORD, TWO OR MORE CASES

Issues of 1906-1915, carried to 1920, 608 cases. Mortality about 5 points below the general average.

## RENAL COLIC, GRAVEL OR CALCULUS

536 policies, issues 1901-1908, carried to 1915, and 785 policies, issues of 1906-1915, carried to 1920; in both periods the mortality was slightly less than the general mortality of the company.

## RHEUMATISM, ACUTE ARTICULAR (MUSCULAR RHEUMATISM EXCLUDED)

1,780 policies, issues of 1906-1915, carried to 1920—68 deaths. Mortality slightly below the general average of the company.

## MALARIAL FEVER, REMITTENT

1,003 policies, issues of 1885-1908, carried to 1915, 59 deaths. Mortality a few points below the general average of the company.

## MALARIAL FEVER (OTHER THAN PERNICIOUS AND INTERMITTENT)

17,602 policies, issues of 1885-1908, carried to 1915, 1,192



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deaths. Mortality slightly below the general average mortality of the company; and 8,975 policies, issues of 1906-1915 carried to 1920, 335 deaths. Mortality about general average of company.

### PRINTING BUSINESS

We issued 3,192 policies during the years 1885-1908, carried to 1915, covering persons engaged in the following branches: Hand Compositors; Linotype Operators; Pressroom Employees; Stereotypers; Electrotypers; Photo-Engravers and Lithographers. Mortality much below our average mortality.

#### COMPOSITORS (JOURNEYMEN)

During the years 1906-1915, carried to 1920, we issued 1,757 policies, with 45 deaths and a mortality of 62.27 per cent of the American Men Table.

#### PRESSMEN (JOURNEYMEN)

352 policies were issued—covering the same period—with 11 deaths and a mortality of 52.48 per cent of the American Men Table.

(We have been unable to select exceptional cases in the following classes which will produce a satisfactory mortality.)

#### IRITIS RHEUMATIC OR OTHERWISE

187 cases, issues of 1885-1908, carried to 1915; the mortality was almost double the general average of the company.

#### PROPRIETORS AND SALESMEN IN AUTOMOBILE STATIONS AND GARAGES

We issued 1,480 policies during the years 1906-1915, mortality carried to 1920, and there were 49 deaths, with a mortality of 151.33 per cent. American Men Table.

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### REPAIRMEN AND MACHINISTS IN AUTOMOBILE STATIONS AND GARAGES

We have issued 651 policies—covering the same period—with 22 deaths, and a mortality of 142.17 per cent.

### CHAUFFEURS, NON-RACING, NOT INCLUDING TESTERS AND DEMONSTRATORS

517 policies were issued—covering the same period—with 19 deaths and a mortality of 169.81 per cent.

### GALL STONES OR HEPATIC COLIC

393 cases, issues of 1885-1908, carried to 1915; mortality was more than double the general average of the Company.

### GASTRIC AND DUODENAL ULCERS

We have had but little experience with persons suffering from the above.

Dr. Hobbs—Any further remarks in regard to this paper. I really believe the paper is an important one to us all. We are all brought face to face with insurance records and the question arises in our minds: What value shall be placed upon these records? I find in Dr. Rockwell's paper the interesting notation that more attention was paid to circulatory diseases and to urinary diseases than to any others. I think this would naturally be so. In regard to the other diseases, the impairments mentioned, the percentage is considerably smaller. I think there is a big difference in the practice of the different insurance companies as to just what bearing the record has upon their actions. I think if we could be more uniform in our consideration of these records and what value we should place upon them; and if the action we take was more in conformity with some general idea as to what should be taken, I think it would benefit the insurance business. This is one of the objects I had in mind when I asked Dr. Rock-

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well to cover this subject, that is, I deduced the fact that a great many companies paid very little attention to records of any kind. I say of *any* kind, of the *peculiar* kind that comes before us all. Other companies pay a great deal of attention to the record while others straggle in from one end of this road to the other.

If we can only have some idea formed in our mind as to what we should do with these records, how much bearing they should have upon the selection or risks, I think we would be very much benefited in our selection. I think Dr. Rockwell's paper has brought out the proportionate number of cases which are influenced by the record. I think we may look over our own action and see whether or not we are acting in conformity or whether we are departing from it or whether we consider that we should pay more or less attention to the records than he does or we have done in the past.

If there is an expression of opinion further than this, I would like to hear from some of the other companies, especially those companies which may be doing a substandard business.

If there is no further discussion, we will pass on to the presentation of the next paper, which is a practical survey of the newer chemical urinary tests. I thought it was time that we got together in regard to some standard urinary test, and having found a workable one, that the different companies should adopt such a test and any report they make should be made thereby. I asked Dr. Muhlberg if he would take this subject up. He has gone very carefully into it. He has issued questionnaires to the different companies' medical departments and he found that the sulphosalicylic acid test for albumin is now, I think, used by 48 per cent of the companies. In my own mind, this test is one of the most practical tests we have for albumin. In the insurance laboratory it comes

nearer to what we want, nearer to what we can easily handle. It may have some objections, but I think they will be ironed out. Perhaps some of these tests will be modified along better lines, but at present, this test is one of the best that we can use and I would advise all the companies to read very carefully the article by Dr. Muhlberg, because it gives information not only about the test itself and the apparatus but how they can install it in their laboratories and so fall in line with what the majority of the companies are doing. Dr. Muhlberg.

Dr. Muhlberg—It is not my intention to read this paper, because it is rather technical and not of very much interest to 48 per cent of you because you are already practising these methods. I would, however, like to offer an apology to the Urinary Committee who may think it somewhat presumptuous on my part to prepare a paper of this sort. My intention was simply to encourage and stimulate the other 52 per cent, who were not using this test to adopt the quantitative methods. I have found that the tests are very readily made and require not much more technical skill than the older tests, and since the greater part of the difficulty in connection with the preparation for doing this work has been now solved by the manufacture of standards for both albumin and sugar tests, I think if you care to adopt the method as practiced by our company, you will find very little difficulty in training your laboratory technicians in doing this work.

I should judge that 60 per cent of the difficulty in connection with adopting the new quantitative test lay in the preparation of the standards. I believe the Metropolitan Life Insurance Company have made some permanent standards. We have been working on permanent sugar standards for the last two or three years and through the courtesy of the Taylor Instrument Company, we have now made available for you at a reasonable price—I don't know just exactly what they are going to charge—five standard tubes according to our

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formula, and they seem to be very permanent. We have had tubes of this sort in our laboratory for two years under all sorts of conditions, exposed to sunlight, kept in dark places, etc., and have found no change in the color of these tubes at all, and I have no doubt that the tubes made by the Taylor Instrument Company will, likewise, be very permanent.

I understand, too, that Dr. Kingsbury has interested some manufacturing house in the project of making his albumin tubes. We have been using Dr. Kingsbury's tubes for about a year and they have proven very satisfactory. One or two tubes have been faulty, but that was due to our technique rather than to the instructions that he gave us. I think you will find that, after you have been using this method for a while, you will be much better satisfied than with the old test. It may be true that quantitative values don't give you all the information that quantitative figures for other impairments do, but there is no doubt of the fact that if we want to select our risks scientifically, we must have quantitative values, and I think actuarial studies will show that the mortality will bear some relation to quantitative values. I have no doubt at all that a group showing  $1/2$  per cent of sugar will develop a lower mortality than a group showing 2 per cent of sugar for the simple reason that those cases showing  $1/2$  per cent of sugar will include a larger number of physiological glycosurias.

We have been very much interested in our laboratory in working on the subject of urinary preservatives, and I thought it would not do any harm to discuss that question in a few paragraphs. The preservative that we use, urotropin and salicylic acid, has certain objections, but on the whole works very well indeed. Urofix tablets are quite satisfactory, but we found through careful research that oxy-gemoline, which can be used in smaller tablets, are quite as satisfactory as the Urofix tablets.

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With these few introductory remarks, I will be very much interested in listening to the discussion.

### A PRACTICAL SURVEY OF THE NEWER CHEMICAL URINARY TESTS

BY DR. WM. MUHLBERG

*Medical Director, Union Central Life Insurance Co.*

Pres. Angier B. Hobbs suggested that it might be of interest to members of the Association to have a paper submitted, detailing the technique and experiences of some one company employing the newer quantitative albumin and sugar tests. He further suggested that a questionnaire be circulated amongst the regular members, with a view of ascertaining how many of these companies were using the tests, with what modifications, etc.

I accordingly sent the following list of questions to our regular members:

Have you a laboratory for the microscopical and chemical analysis of urine at your Home Office?

If so, what tests are you using for the detection of albumin?

For the detection of sugar?

If you are not using the Benedict Picramic Acid Test or the Folin or Exton's Sulphosalicylic Acid Quantitative Albumin Test or one of the modifications, are you planning to do so in the near future?

Are there any reasons why you have not as yet decided to adopt these methods?

Do you believe that a paper detailing the exact methods employed, the preparation of the standards and acquiring and installing the equipment, etc., would be of interest to you at this time?

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Do you believe that our examiners in the field should be instructed to use these tests for routine examinations?

What chemicals are you using for preserving the samples mailed to your laboratory?

Have you found this preservative entirely satisfactory?

Would a discussion of urinary preservatives be of interest to you?

The replies are interesting, in that they indicate that of seventy-three companies responding—

48% are using the Quantitative Albumin Test. This represents 83% of the total amount of insurance in force carried by the 73 companies answering the questionnaire.

36% are using the Quantitative Sugar Test. This represents 46% of total amount of insurance in force.

30% contemplate doing so.

48% are using one or both tests.

22% are satisfied with the old methods.

80% were of the opinion that a paper detailing exact methods would be of interest.

Practically all companies were opposed for the present to instructing the examiners in the field to use the tests.

With reference to preservatives—

29 used Urofix Tablets	40%
15 used formaldehyde	20%
20 used boric	27%
9 used miscellaneous preservatives	13%

Practically all felt that the ideal urinary preservative had not yet been discovered.

It is quite evident from letters written as supplementary to the questionnaire that many companies are hesitating to adopt the newer tests, not because of any lack of confidence but rather because the technique is somewhat complicated, the standards not easy to make and because our experts are

still in controversy over some of the details. It is, therefore, evident that a purely practical paper, describing in detail how some average laboratory switched over from the old to the new methods, would not only be of interest, but would be of assistance to such companies as have not as yet taken the plunge. Such a paper, to be of any value, must necessarily be rather elementary and dogmatic and must avoid taking issue on controversial points.

It is only with great reluctance that I have undertaken this task and I am doing so only because I feel that the newer methods, even if not as yet perfected, are probably better and more accurate than the old, are easily practiced, and if once inaugurated can be used as the basis for future changes or improvements. I do not pretend that our methods are the best, nor that they are free from criticism, but I do believe that they are so satisfactory that we would not consider reverting to the older technique. Besides, unless a quantitative method is employed, it is difficult or impossible for any company to report their findings accurately through the interchange bureau.

It must be remembered that while quantitative estimations of sugar and albumin are important in deciding the significance of the impairment, these quantitative values are by no means as informative as are such factors as blood pressure in millimeters, or of exact height and weight, etc. An applicant with only a trace of sugar may be a potential or even a real diabetic, whereas even large amounts of sugar may represent merely a dietetic error or a reaction to nervous excitement; and this likewise holds good for varying amounts of albumin. The quantitative reports must be correlated with other factors, such as previous history, build, blood pressure, casts, family history, etc.

We have been very fortunate in securing for our laboratory the services of a chemist who has worked as



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an assistant in the laboratories of Professor Benedict. She had first-hand knowledge of the newer methods, introduced the technique, prepared the standards and instructed our laboratory force. I believe that it well repays any company doing Home Office laboratory work to employ an expert, at least part time, if for no other purpose than to keep the laboratory methods up to date. We are really greatly indebted to the research studies of the experts who have been working in the laboratories of the larger companies. They have not only modernized the methods and improved our technique, but have jolted us out of a self-complacency that might eventually have meant stagnation.

We have so far as possible, tried to adhere to the recommendations of the Committee on Urinary Impairments, as reported at the Thirty-fifth Annual Meeting of the Association, and I fully concur with the conclusions of Dr. Patton in his comments on this report.

### QUANTITATIVE TEST FOR ALBUMIN

#### I. *Reagent*—

3% sulphosalicylic acid solution. This can be prepared by any druggist, but the sulphosalicylic acid should preferably be purchased from some reliable manufacturer—such as Eastman Kodak Co., Rochester, New York.

#### II. *Apparatus*—

(a) Special test tubes, each one graduated to 2.5 and 10 cc. These tubes must be of uniform diameter and carefully graduated. They can be obtained from the Emil Greiner Co., New York.

(b) It is convenient to keep the sulphosalicylic acid in a bottle which has a side opening near the bottom (Quart aspirator bottle with irrigation attachment). To this opening is attached a bent glass tube, rubber tube with pinch cock

and glass tip, so that the sulphosalicylic acid can be drawn directly from this into the test tube.

(c) Set of standards whose turbidity corresponds to the turbidity given by solutions of albumin containing 5, 10, 20, 30, 40, 50 and 100 mg. of albumin per 100 cc. Directions for making these permanent standards according to Dr. Kingsbury, are as follows:

"Dissolve fifty grams Super X Brand Gelatin Sheets, manufactured by Coignet Chemical Products Company, New York, in three hundred cc. water fifty degrees centigrade; make up to five hundred cc. Stir in white of one egg and heat for half hour after reaching ninety degrees. Filter hot. This is the clarified ten per cent gelatin medium for standards. Call this gelatin stock. Make Formazin as follows: Dissolve one-quarter gram hydrazine sulfate from Eastman Kodak Research Laboratory in twenty-five cc. water. Dissolve two and one-half grams Urotropin in twenty-five cc. water and add whole of this solution to whole of hydrazine sulfate solution. Allow mixture to stand in stoppered flask over night. This is the Formazin Suspension. Make one hundred milligram standard by adding fourteen and one-half cc. of Formazin Suspension, inverting thoroughly to mix one hundred cc. melted gelatin stock. Mix to make uniform suspension. Call this 'A.' Use twenty-five cc. 'A' and twenty-six cc. gelatin stock for fifty milligram standard; twenty 'A' and thirty gelatin stock for forty milligram; fifteen 'A' and thirty-five gelatin stock for thirty milligram; ten 'A' and forty gelatin stock for twenty milligram; five 'A' and forty-five gelatin stock for ten milligram standard. Add one drop, no more, forty per cent formaldehyde to each ten cc. completed standard, invert to mix, stopper and allow to cool and keep fairly cool for few days. One hundred milligram standard equals one-tenth per cent albumin."

#### DIRECTIONS FOR THE METHOD

The urine to be tested is well centrifuged and the clear urine is introduced by means of a pipette into one of the graduated test tubes to the 2.5 cc. mark. The tube is then

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filled to the 10 cc. mark with 3% sulphosalicylic acid inverted to mix, and allowed to stand for 10 minutes. At the end of this time the turbidity developed in the tube is compared with the turbidity of the standard tubes, to find out which standard it most nearly matches.

The comparison is most easily made by holding the tube and one of the standards side by side at a little below the level of the eye, against the light from a window, and then holding a white paper, on which black lines of varying heaviness have been drawn, obliquely behind the tubes and observing the appearance of the black lines as seen through the turbidity of the tubes. By using this method of comparison the interfering effects of different colors, and of side lights seem to be cut to a minimum.

The amount of albumin in the urine quantitatively equals the standard tube whose turbidity it most nearly matches.

The sulphosalicylic acid method tends to give lower results than the heat and nitric acid tests, due to its greater specificity for albumin. There are, however, certain substances like spermines, protamines, etc., which may react with the sulphosalicylic acid. The turbidity given by most of these substances disappears when the tube is warmed. (See Exton, W. G., *A Simple and Rapid Quantitative Test for Protein in Urine*. J. Lab. and Clin. Med., x, 1925, p. 3.) Where there is any doubt it is well to warm the tube to see whether it clears up. The precipitate formed by albumin itself does not change with warming.

Cloudy urines are difficult to estimate quantitatively, hence the necessity of using a good preservative.

Dr. Chas. P. Clark, of the Mutual Benefit, has devised an excellent albuminometer, which we purchased through him from the manufacturer, Mr. Kenworthy. We have been using this albuminometer in our laboratory and have found it very satisfactory; in fact, we are inclined to rely entirely upon it,

because more consistent results are obtained from day to day, where a uniform lighting system is used.

Dr. Exton has made available through Lehn & Fink, of Bloomfield, N. J., and at a price of \$10.00 a complete outfit of standards, albuminometer and reagents, with full directions for their use. We have been using Exton's method side by side with Kingsbury's and have found that both methods coincide very closely, with a slight tendency on the part of Exton's method to read higher quantitatively than Kingsbury's. (The choice between the two methods is mostly a matter of personal opinion and of familiarity with the technique.

In our work, we have found that neither Kingsbury's nor Exton's albumin standard tubes are really permanent. In one or two of Kingsbury's tubes, slight mold developed, and in some of Exton's tubes the particles become coarser or more granular with age. But this difficulty can be overcome by replacing with new standards when deterioration develops. I rather suspect that the mold that developed in the Kingsbury tubes was due to some fault in our technique in preparing them.

#### BENEDICT'S CLINICAL QUANTITATIVE TEST FOR SUGAR IN URINE

##### *Reagents—*

0.2% picric acid solution.

5% sodium hydroxide solution.

50% acetone (must be made fresh each day by dilution of one part of acetone with an equal volume of water).

##### *Apparatus—*

(a) Special test tubes of equal diameter, graduated to 25 cc. These can be obtained from the Emil Greiner Co., New York.

(b) 5 cc. special pipettes, graduated in 1 cc. also obtained from the Emil Greiner Co., New York.

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(c) Test tube racks, of size to hold tubes.

(d) Water bath, with rack to hold tubes to fit it. A copper water bath, with tripod and a convenient test tube rack to fit the bath may be purchased for \$8.10 from Max Woche & Son Co., 29 West Sixth Street, Cincinnati, Ohio.

(e) It is also convenient, to have burettes from which to measure the picric acid and the sodium hydroxide. 50 cc. burettes are appropriate for the former and 25 cc. for the latter. Since 0.5 cc. portions are to be measured from the latter, it is best to have the graduations spaced as far apart as possible. It is also convenient to have a dropper-bottle for the acetone solution.

### *Directions for the Method—*

By means of the special pipette, measure 1 cc. of urine to be tested and introduce into special test tube. Add 3 cc. of 0.2% picric acid solution from burette, and 0.5 cc. of 5% sodium hydroxide from burette. Then add by means of dropper-bottle 5 drops of 50 per cent acetone (prepared fresh each day by dilution of one part of acetone with an equal volume of water). These reagents must be added in the order named. Transfer the tube immediately to the water bath and heat for 12-15 minutes. Remove the tubes, cool, dilute with water to the 25 cc. mark and mix. The color of the solutions thus obtained indicates the amount of reducing sugar present, up to a concentration of 0.5%. The colors of the solutions are then compared by transmitted light with the colors of the standard solutions which are kept in sealed tubes of the same diameter as the tubes used in the determination. The colors of the standards correspond to those obtained from 1 cc. of 0.1, 0.2, 0.3, 0.4 and 0.5 per cent of pure glucose treated in exactly the same way as the urine.

In the use of this method it should be noted that the quantity of picric acid present during the reaction is not suffi-

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cient to react with much more than the glucose present in 1 cc. of a 0.5 per cent solution. Therefore, urines containing larger quantities of sugar may not give much more color than those containing 0.5 per cent and such urines should be diluted and the determination repeated.

### DIRECTIONS FOR MAKING PERMANENT STANDARDS TO BE USED WITH BENEDICT'S CLINICAL QUANTITATIVE TEST FOR SUGAR IN URINE

#### *Chemicals used:*

(It is important that chemically pure products be used.)

*Potassium bichromate*, C. P. Baker's Analyzed.

*Chromium Potassium Sulphate*, "Chrome Alum"—Eimer and Amend Tested Purity.

*Cobalt Chloride*, C. P. Baker's Analyzed.

*Sulphuric Acid*, C. P.

#### *Directions:*

*0.5 per cent Standard:* 8 gm. potassium bichromate, dissolved in water with 6 cc. concentrated sulphuric acid. Cooled. 0.6 gm. chrome alum added and dissolved. Made up to 100 cc.

*0.4 per cent Standard:* 6.66 gm. potassium bichromate, dissolved in water with 5 cc. concentrated sulphuric acid. Cooled. 0.55 gm. chrome alum added and dissolved. Made up to 100 cc.

*0.3 per cent Standard:* 4.6 gm. potassium bichromate, dissolved in water with 5 cc. concentrated sulphuric acid. Cooled. 0.5 gm. chrome alum added and dissolved. Made up to 100 cc.

*0.2 per cent Standard:* 0.52 gm. potassium bichromate, dissolved in water with 1.5 cc. concentrated sulphuric acid. Cooled. 0.1 gm. chrome alum and 3.2 gm. cobalt chloride added and dissolved. Made up to 100 cc.

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*0.1 per cent Standard:* 0.075 gm. potassium bichromate, dissolved in water with 1.5 cc. concentrated sulphuric acid. Cooled. 0.172 gm. chrome alum and 1.48 gm. cobalt chloride added and dissolved. Made up to 100 cc.

Tubes suitable for containing the standards can be obtained from the Emil Greiner Co., New York. These tubes must have the same diameter and be made of the same glass as those used in the sugar determination, but they can be obtained without the graduation to 25 cc. which makes them less expensive. The tubes are first drawn out at the end, by means of a hot flame, the liquid is introduced through the small opening, and the tubes are sealed off so that they are perfectly airtight. If exposed to the air the solutions darken.

Since our artificial standards match the original standards perfectly when the two solutions are compared by the colorimeter, it is immaterial whether natural or artificial light is used.

The Taylor Instrument Companies, of Rochester, New York, have very kindly consented as a courtesy to the members of this Association, to manufacture our sugar standard tubes according to our formula. These may be purchased from them at a reasonable price.

The Metropolitan have likewise devised a set of permanent standard tubes, but we have not had the opportunity to work with them in our laboratory.

It is to be hoped that Dr. Kingsbury will induce some reliable instrument maker to manufacture and make available, his albumin standards.

### URINARY PRESERVATIVES

During the past two years, we have been experimenting with various preservatives. It is very essential in connection with any quantitative test that an antiseptic be used that not only preserves the urine, but also does not in any way in-

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terfere with the reactions. Bacteria growing in the urine cause a turbidity that makes it almost impossible to apply the albumin test, and many urinary antiseptics have a reducing action similar to glucose.

The preservatives which were tried out included the inorganic substances, sodium fluorid, zinc chlorid, boric acid and borax; the organic substances, formaldehyde, toluene, thymol, trikresol, resorcinol, hexamethylresorcinol, benzoic, salicylic acid and their salts, quinoline, oxyquinoline, camphor, menthol, helmitol, acriflavine, chlorazene and the Urofix tablets.

Our conclusions based on painstaking research with normal and infected samples, exposed to laboratory and transportation conditions, and studied with samples at room and incubator temperatures, may be of interest.

Formaldehyde in glass tubes is unquestionably the best preservative. There is, however, the major objection that it requires glass ampoules, which are breakable. It is furthermore, not fool-proof. It slightly reduces Fehling's solution and the Benedict's picramic test and when too concentrated forms globules that interfere somewhat with the microscopic examination.

The next best preservative is a powder, consisting of three parts of hexamethylenamine with two parts of salicylic acid, intimately mixed. 200 mg. or a little over 3 grains of this powder are placed in the bottle with a measuring cup. The bottle and cork must be dry. The mixture evolves formalin when moistened with urine and the sample is preserved as well as when formaldehyde is used. No deterioration, chemically or microscopically, occurs after weeks of incubation. The objection, of course, is that the mixture is a powder, which the examiner, unless warned, may throw out; and since salicylic acid is used, it interferes with the Ferric chlorid test for diacetic acid.

Oxyquinoline or chinosol ranks next best. Used as a pow-



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der, in amounts of one grain, it preserves the samples beautifully, but gives them a yellowish tinge. It likewise gives a bluish color with Ferric chlorid.

Of the tablets, three commend themselves: First, Dr. Kingsbury's tablets, made according to his especial formula. Secondly, tablets of hexamethylenamine and salicylic acid, made by Sharp & Dohme according to our formula; and finally, Urofix tablets.

The Urofix tablets apparently contain oxyquinoline and alum. They preserve the urine very well, tinge it yellow, often form slight brownish sediments and possibly on account of the alum, they interfere slightly with the quantitative albumin test.

Boric acid cannot be recommended at all. It is not a very good preservative, it interferes with the picramic acid test, and yeast grows very freely in boric acid samples.

We make a practice of sterilizing by boiling our bottles and corks and this practice is recommended, inasmuch as it adds at least fifty per cent to the effectiveness of any preservative.

Dr. Hobbs—Drs. Folin and Benedict were expected to be here this afternoon. Dr. Folin, would you rather at this point make a few remarks or wait until after the discussion?

Dr. Folin—We are here essentially as listeners to hear what the real practical workers have to say and we would rather listen to the others for the present.

Dr. Hobbs—In the discussion, we will hear first from Dr. Clark of the Mutual Benefit.

Dr. Clark—Mr. President and Gentlemen: Dr. Muhlberg has presented fully the technical points incident to the employment of the quantitative tests for albumin and sugar. It might be well for me, in the time allotted, to give a statement of the practices of the Medical Board of our company

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in the interpretation of the reports obtained by these tests, and our experience with the tests.

### THE QUANTITATIVE ALBUMIN TEST

I might state primarily that our Board is unanimous in the opinion that a report of .005% is in many cases of great significance, and we should not discontinue the use of that standard. When the amount of albumin is less than .005%, its presence, we believe, may be ignored. Our judgment in this matter has been developed after using this test continually for over two years.

(2) The Quantitative Test gives more dependable information than either the Heller's Ring Test or the Heat-Acetic-Salt Test, because it eliminates almost wholly the personal equation, which is an important factor in the interpretation of results with the latter tests.

(3) It has been possible, because of the adoption of this test, to liberalize in our selection in that group of cases showing the presence of albumin, particularly in small amounts. We are hopeful that we may go further in this respect.

(4) From the laboratory viewpoint we have found the test simple, rapidly performed, and it has been from the beginning conducted by a lay technician without previous laboratory training.

(5) The standards which we are now using were made 18 months ago and are unchanged. When changes have occurred, they may now be replaced at a low cost.

(6) The lamp which Dr. Muhlberg mentioned has proved of great value in our laboratory where daylight is not always available and where the quality of daylight is ever changing.

(7) Up to the present time we have been using the Heller's Ring Test as a supplementary test. I do not feel personally

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that this supplementary test has been of any great advantage to us, inasmuch as our underwriting decisions are based wholly upon the report of the Quantitative Tests.

(8) When spermatic elements are found in the microscopical sediment, we routinely heat the test tube containing the precipitated albumin and report the finding after heating. In some cases we have had decreased turbidity, as much as 80%.

(9) We have supplied the offices of our New York and Chicago agencies with complete outfits and both agencies feel that the test is superior to the older tests and they do not care to revert to the use of the former. However, as a general practice we do not feel that it is absolutely essential that all agencies be equipped with the outfit, inasmuch as all specimens in which the examiner finds albumin must be sent to the Home Office for further examination.

### QUANTITATIVE SUGAR TEST

We have employed the Picric Acid Quantitative Sugar Test devised by Dr. Benedict with the standards developed by Dr. Kingsbury for over a year and our Medical Board agree that the test is of great value in our medical underwriting. We are, however, using the Haines Qualitative Test as a supplementary test. If from these tests we have positive or suggestive evidence of glycosuria, the specimen is examined also with the Benedict Qualitative Test and the Fermentation Test. Quite frequently our judgment of the merits of the case is dependent upon the combined results of all four tests, bearing in mind the specific gravity of the specimen and the collateral evidence at hand. The Medical Board agree that we should not change this system although it entails additional work in our laboratory. We feel that by receiving all four reports we are in a better position to know how far we can liberalize in our treatment of the applicant himself.

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At the meeting of the American Life Convention Dr. Kingsbury and I presented tables showing the results obtained by our two laboratories covering many thousand specimens. These tables are contained in our reports to the American Life Convention which most, if not all, of you have available. We agreed that the amounts of sugar below .2% may be ignored. A report indicating .2% or .3% sugar, particularly in a specimen with low specific gravity, is suggestive of a glycosuria. When the amount present is .5% or more, we believe that we are dealing with a true glycosuria. I have found that in the great majority of our cases in which one or more specimens have shown the presence of .5% or more of sugar, we have declined or postponed. Three or more specimens have been examined in these cases and all factors presented by our examiner, together with the M. I. B. reports have been considered. These cases apparently are for the most part sub-standard.

In our laboratory there has been no embarrassment in conducting this test which has been from the first carried out by a lay technician. The standards which Dr. Kingsbury kindly gave us a year ago appear to be unchanged at this time.

### PRESERVATIVES

We have made, up to the present time, no change in our method of preserving specimens. With each mailing case and bottle is sent a small vial containing 2 c. c. of a 1/2% formaldehyde solution. The cork is covered with paraffin to prevent evaporation. There are several disadvantages in employing this preservative. The time required in our laboratory to prepare these vials is considerable and must be taken into account in considering the cost. The preservative properties of formaldehyde are well known to be excellent, but we believe from our experience that many of our decomposed specimens

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are due to the fact that although the examiner has properly preserved the specimen at the time of voiding he has not been able to make his own examination until one or two days have elapsed. The specimen is then examined by him and no further preservative is added. Sufficient formaldehyde has escaped after opening the bottle so that an inadequate amount of formaldehyde is retained in the specimen and by the time the specimen has been received at the Home Office it has decomposed. I have had personally so many experiences indicating the necessity of adding at least another drop of formaldehyde, when the preserved specimen is thus examined and then mailed that I feel that our percentage of decomposed specimens is greatly increased by the lack of proper technique on the part of the examiners. Therefore a tablet in which the preservative is fixed and which will not evaporate would be very superior to formaldehyde used in the way in which we are now using it. I believe on the whole it would be less expensive to the company if we were to employ such tablets. I have not yet had the opportunity to fully test the value of the tablets designed either by Dr. Wolfe or by Dr. Kingsbury. However, from my observation of the specimens preserved with these respective tablets in both the Equitable and the Metropolitan laboratories, I would be led to believe that they are both quite dependable.

In conclusion I wish to call attention to the sentence in Dr. Muhlberg's report to the effect that the research studies have jolted us out of a self-complacency which might eventually have meant stagnation. It is a frequent fault of all institutions to continue the use of methods which are long out of date either through lack of attention to details or through prejudice. In the fields of chemistry and physics many improvements have been made which are available to the laboratories of the insurance companies and we should be ever alive to the utilization of these important improvements.

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Furthermore, contrary to the usual belief, it is my impression that such methods are not only advantageous to the insurance companies employing them but also to the insuring public.

Dr. Hobbs—Dr. Kingsbury.

Dr. Kingsbury—Mr. President and Gentlemen: In discussing Dr. Muhlberg's paper, it seems necessary to bring out a few points. Dr. Muhlberg, I think did extremely well to make our gelatin standards from the meager information contained in one telegram. Two of his standards became mouldy. This was due I thought at the time to a lack of formaldehyde, but after a similar occurrence in our laboratory when special precautions had been taken to make sure that each tube got sufficient formaldehyde I was quite at sea about it, for we had made more than one thousand standards before this happened with three only showing any signs of decomposition.

At any rate, the Fales Chemical Company of Cornwall Landing which through an arrangement made by Dr. Knight is undertaking the manufacture and distribution of these standards as well as the permanent sugar standards and our preservative tablets, will make reasonably certain that the albumin tubes will not decompose, before distributing them. I might add that the Fales Chemical Company is working in closest co-operation with our laboratory in these matters.

These formazin albumin standards are permanent for eight months to a year. The standards which Dr. Clark referred to as lasting for eighteen months are of a type which is difficult to make and difficult to describe for anybody else to make. We have some that are now two years old and perfect, but prefer the formazin standards on account of the simplicity of preparation.

Our experience with Dr. Exton's standards agrees with that of Dr. Muhlberg. They give close agreement with our stand-

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ards when new, but after ageing a few months the turbid material precipitates and the tubes of lower value are rendered worthless.

We had occasion to make some permanent sugar standards according to Dr. Muhlberg's directions and found that they agreed absolutely with our own for the corresponding sugar levels. A certain company sent us a set of sugar standards purporting to have been made according to Dr. Muhlberg's directions. They were failures, but they were not made according to Dr. Muhlberg's directions, but according to the ideas of the local druggist. I bring this matter to your attention merely to show how easy it would be to ruin a perfectly good method by having the standards for the method prepared by some one not qualified for this work.

I had the pleasure of discussing a paper of Dr. Muhlberg's on preservatives at Louisville in June, 1925. At this meeting I presented the formula for a preservative which had given good results in the laboratory and up to that time good results in the field. Shortly after I returned from the meeting I was confronted with the incipient failure of the preservative which I had just presented to you as good. More than a year later I can say that in our system of bottle distribution in which the examiner may get a bottle a year or two old this first preservative which I described has got progressively poorer as time has gone on. In the meantime, however, we have modified our formula and have had a year's experience with our last formula so that I can say with more confidence that our present preservative which we call by its serial number, 4, is good and I shall present a little comparative data presently to support this. We have made about 150,000 tablets up to this time and have just recently turned over the manufacture and distribution to the Fales Chemical Company, previously mentioned.

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### Preservative No. 4

#### Per Tablet for One Ounce Urine

Potassium acid phosphate	0.100 gram
Sodium benzoate	0.050 "
Benzoic acid	0.065 "
Urotropin	0.050 "
Sodium bicarbonate	0.010 "
Mercuric oxide, red	0.001 "
	<hr/>
	0.276 "

Briefly our experience for eleven months is that with No. 4 the percentage of decomposed specimens in the hottest months of the year has never been over 3.5 and in the cooler months runs as low as 2. With "Urofix" the average is about 5 per cent and with boric acid anywhere from 5 to 10 per cent, depending upon the season of the year.

One of the chief qualities that a preservative for urine must possess is that of keeping the sugar up to its original value. No. 4 does this very well in most cases for five days at room temperature and much longer in certain cases. Dr. Connolly of our laboratory has shown that red blood cells are maintained in their original state and number for as long as four months at room temperature in three out of four urine specimens and more than a month in the fourth specimen preserved with No. 4. Leucocytes are also preserved for long periods. Casts are preserved well at least a week at room temperature, much longer in some specimens.

Dr. Clark in his discussion has called attention to the fact that a specimen showing spermatozoa in the sediment may give an entirely erroneous test with sulfosalicylic acid. He has also shown the remedy, heating the test, when the false turbidity disappears leaving that of the albumin-globulin mixture if these substances are present. This is also our practice



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and I shall add only the caution that the test tube should be heated to 50 centigrade and not merely waved through the flame once or twice, as slight warming of this kind does not dissolve the turbidity caused by the proteose-like compounds present in specimens of this kind.

In closing I might add that we have made literally hundreds of parallel determination of urine specimens at all levels of sugar content with the Benedict picrate test and two well-known and accurate quantitative copper methods which were applied to the same urines only after interfering substances had been removed and that the Benedict picrate test gave accurate results in terms of the other methods at all levels of sugar where glucose itself was present in any predominating proportion of the total urinary sugar, in other words at the levels which are of any possible pathological significance and therefore of significance in insurance work.

I wish to thank Doctor Hobbs for inviting me to discuss Dr. Muhlberg's important paper on quantitative methods.

Dr. Hobbs—Dr. Exton.

Dr. Exton—May I say to the 52 per cent of companies who do not yet use the quantitative test that I recommend as a reasonable proposition that you run along with the tests you are now using, the quantitative tests a few hundred times. I think that if you will do this, no further arguments will be needed to persuade you or to convince you that from the standpoints of both laboratorian and medical director who is acting on the case, that the quantitative tests represent a real and great advance in our laboratory work. Dr. Muhlberg and some of the other speakers have mentioned some points connected with the use of the sulfosalicylic test which deserve comment. Thus Dr. Muhlberg touches on the matter of the cloudy specimens. I do not advise wasting time to no purpose on decomposed specimens, but there are always a certain number of specimens which are not decomposed but

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which are, nevertheless, cloudy. Attempts should be made to clear such specimens by bringing the phosphates or urates, as the case may be, into solution. Even with this procedure and centrifuging, some specimens will still remain cloudy either because of bacteria or some less usual cause. The sulfosalicylic test used in our laboratory enables us to get quantitative results on such cloudy specimens without difficulty. All you have to do is to add an equal volume of water to the cloudy urine and read the cloudiness in the scopometer. Then take another sample of the urine and apply the sulfosalicylic test in the usual way and read the result. The difference between the readings of the two clouds thus produced will give the correct quantitative result. There is nothing to add to what previous speakers have said about standards for the albumin test because for more than a year now the matter of standards has been to us merely of academic interest. Our laboratory was the first to make and use permanent standards, but we have discarded them because we find the scopometer method is better in every way. I was glad to hear all of the speakers touch on the matter of warming the sulfosalicylic acid test. As I originally pointed out, every test should be warmed. If you rely on finding spermatozoa with the microscope you are misreporting other cases in which the cloudiness is due to other substances than either spermatozoa or albumin. We have seen many such cases in women and people who take resinous and other kinds of medicine. They give clouds which disappear on warming the test, and only by warming them will you escape making false reports. In my opinion, the specificity of sulfosalicylic acid for the proteins we are interested in, is its greatest advantage.

With regard to sugar tests, let us keep clearly in mind that they involve two distinct considerations: First, the finding of actual sugar, and, second, the matter of what the significance of a given finding of sugar may be. As Dr. Muhlberg

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discusses only the matter of tests for finding sugar, I will also ignore the second consideration with the mere mention that Dr. Anton Rose has worked out in our laboratory a very efficient preservative for blood, and that some such method as Dr. MacCrudden's can be employed for gauging the significance of a glycosuria. The Prudential laboratory still employs the same methods that have been in use practically since the laboratory started, and has always, even from the beginning, reported sugars quantitatively. A Benedict qualitative copper test is made on every urine. We take as a dividing line and as meaning about 2/10 of 1 per cent of sugar a greenish tinge of the precipitate and get such or more pronounced reductions in about 7% of all the urines examined in connection with insurance. Thus 93% of all specimens can be handled with a minimum of time and manipulation. The remaining 7% are either fermented or, if the reduction is very slight, treated with phenylhydrazin. By these means it is aimed to exclude all the nonglucose reducing substances as far as possible in order to avoid complicating selection, because glucose for the present at least may be considered as the only sugar of insurance interest. What is really needed is a simple technique that will be as specific for glucose as possible and at the same time quantitate sugar over the whole range say from .2% to 10%, without putting us to the trouble of repeating tests with different dilutions.

Now just a few words about the very important matter of preservatives. We have been using formaldehyde for more than eleven years and during all this time have been looking for something better and continually trying out other preservatives. Up to the present we have been able to find nothing as satisfactory as is formaldehyde in simple solution. Dr. Muhlberg has mentioned some of the disadvantages connected with formaldehyde in his paper, but it may be said that the preservative which Dr. Muhlberg is using has all of the

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disadvantages of formaldehyde plus other disadvantages which are connected with salicylic acid. On principle it seems to me unquestionable that preservatives should be as simple as possible and that mixtures only lead to complications. We have tried out carefully many of the various tablets and powders that have been proposed for preservatives and have found that some of them raise the specific gravity three or four points. Many of them change the surface tension which, in turn, alters the character of the sulfosalicylic-albumin precipitate and may thus vitiate the result of albumin tests. When using tablets, powders and mixtures these effects should be known and borne in mind and allowed for in reporting results.

In conclusion, I would like to voice our indebtedness to Dr. Muhlberg for the very clear and comprehensive way in which he has handled the subject-matter of his paper.

Dr. Hobbs—Are there any remarks to be made or questions to be asked Dr. Exton? Does anybody want to speak about anything that has been brought out in his demonstration?

Dr. Kingsbury—It has been shown before that one has the fixation effect with large amounts of albumin, but it seems peculiar that one should have a fixation effect with a substance which is not a normal excretory substance at all.

Dr. Hobbs—I would like to hear a word now from Dr. Exton in regard to the paper about the scopometer so that we may also hear from Dr. Folin and Dr. Benedict in regard to this matter.

THE SCOPOMETER—A UNIVERSAL INSTRUMENT  
FOR MEASURING COLOR, CLOUDINESS AND  
OTHER OPTICAL PHENOMENA OF LIQUIDS

Introductory Note

By DR. WM. G. EXTON

*Director of the Prudential Laboratory*

In 1916 the Prudential Laboratory planned and inaugurated an albuminuria program whose objective was the eventual development of possible ways and means of definitely determining the significance of albumin findings in the urines of applicants for life insurance. In short, we have been working toward developing methods that would tell us truthfully the meaning of an albumin finding in a given case in order that acceptance, rating or rejection of a risk can be made efficiently on the merits of the particular case.

The work has been carried along at such times as could be spared from the routine of the laboratory, and it is confessedly with the hope of expediting this program that I depart from the usual custom and speak now of our incomplete work, asking your indulgence in this matter not only because we have somewhat improved our own albuminuria selection, but also because the work has reached a stage which promises practicable methods for selecting albuminurics with definite knowledge of the significance of the albumin found in a given case.

By touching as concisely as possible on a few points connected with the work I hope to make it clear to you that such a program consists of a number of isolated problems which need elucidation and which, therefore, lend themselves perfectly to intensive study in separate laboratories; also that a concerted constructive effort would, in a few years, arrive at a means of selecting albuminurics rationally instead of by ar-

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bitrary office rules. It is therefore suggested that something like a permanent committee on albuminuria might come into being to function with this end in view.

Anyone who contemplates the general objective of trying to find ways and means definitely to determine the significance of an albumin finding in a given case immediately realizes that even by clinicians very little is known about the manner in which the proteins are excreted. Although very important advances in our knowledge of kidney physiology have been made, the ways in which the proteins are excreted are practically unknown. Our first attack, therefore, was to find a way to learn how in different kinds of cases the proteins are excreted.

To illustrate the method developed for the study of this phase of the problem, a few curves, or what might be called pictures of protein excretions, will suffice (slides). Many other such curves or pictures might be shown, but for the present it will be sufficient to say that curves made up to the present show that the manner in which protein is excreted falls into about five different types which correspond with known pathological and clinical entities. This method of typing protein excretion has the advantage of permitting one to classify albuminurics by the type or manner in which the protein is excreted. Such studies are being carried along, and it is hoped that it will have been possible to study sufficient material to present the matter with some degree of finality by next year's meeting.

In connection with what I call typing protein excretion, other studies indicate that by making certain tests on two or three random specimens of urine such as we can get in insurance work it will be possible to gain such knowledge of a case as is given by these curves which portray the protein excretion over a twenty-four hour period. Thus Folin's pioneer work on endogenous metabolism teaches that the creatinine

found in the urine of normal people varies within narrow limits for the individual and that its excretions are but slightly influenced by customary diets. There are other products of endogenous metabolism; of these the uric acid comes from the glandular tissue, while creatinine and neutral sulphur come from muscular tissues. The amounts of creatinine and sulphur excreted are characteristic for the adult, the striated muscular tissue mass and also for the species. In particular, creatinine has been definitely correlated with the total creatine content of the body and in normal individuals on the usual diet is excreted at quite a constant rate. We have, therefore, been estimating creatinine in the specimens examined in connection with typing albumin excretion. The results are promising but much work needs yet to be done. If the necessary data could be worked out in three different laboratories, much time could be saved in gaining the information required concerning these three products of endogenous metabolism. Up to the present time difficulties have been experienced in finding material for study among the milder or intermittent albuminurics, and co-operation or assistance in securing such material would be especially welcome.

The tests we employ for what we call albumin show compositely what is generally regarded as the total urinary protein. As a matter of fact, the protein complex as we see it in our albumin tests is made up of a number of different protein fractions and these and their relations to one another have also been subjects of study. The Prudential Laboratory has developed very simple and rapid methods to estimate separately the albumin and globulin fractions in blood and urine, and our findings thus far suggest that the ratio of albumin and globulin as found in the urine tends to be characteristic.

Another interesting protein feature which needs investigation is presented by the precipitates resulting from application in the cold of mono and dichloroacetic acids to speci-

mens of urine which remain perfectly negative when treated with sulfosalicylic acid. Undoubtedly the mucins account for many of these precipitates, but certainly not for all of them. Involved in this question is the significant matter of nucleoprotein as well as other protein fractions.

I regard as the most promising of all fields of protein investigation the methods used in immunology. Recent publications of Hektoen and his co-workers, Weller and others have advanced the technique, and Gideon Wells has ably emphasized the high degree of specificity of immunological as compared with chemical methods in the study of proteins. With more experience and improved methods for the study and measurement of precipitates the manifest advantages resulting from the far greater specificity of immunological methods may be brought within the realm of the practical.

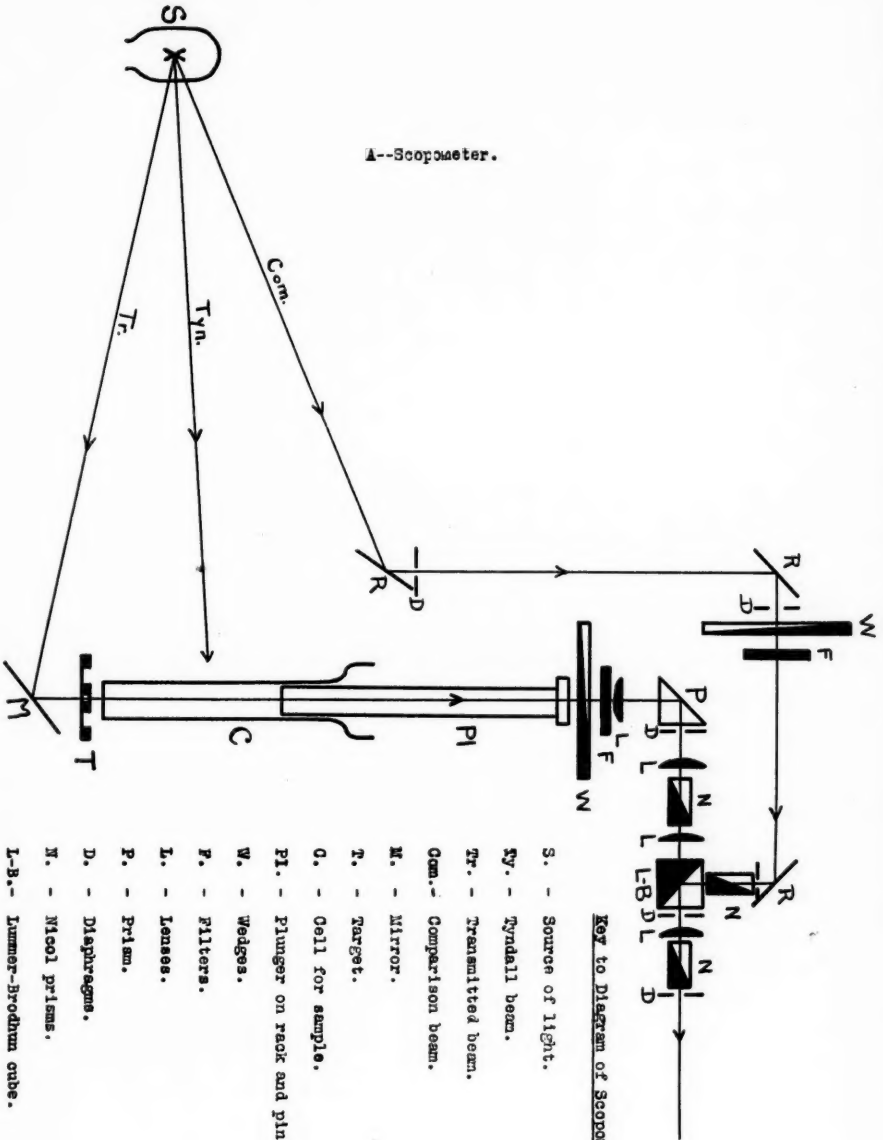
There are many other problems connected with proteins, such as lactic acid, pH, etc., which need to be cleared up, but enough have been mentioned to show that very little is known about these most important substances which hold the keys to so many secrets of health and disease. I may be an optimist, but it seems unquestionable to me that a real effort made co-operatively in different laboratories would soon result in practical means of selecting albuminurics on the basis of the merits of the individual case and that the intensive study of separate problems connected with proteins in different insurance company laboratories would soon yield knowledge of inestimable value, not only for insurance work but also for clinical and especially preventive medicine.

#### THE SCOPOMETER

The nature of the work called for by our program involved the employment of short cut but precise methods for different kinds of analysis and measurement. It was, therefore, not only necessary to improve existing methods which were

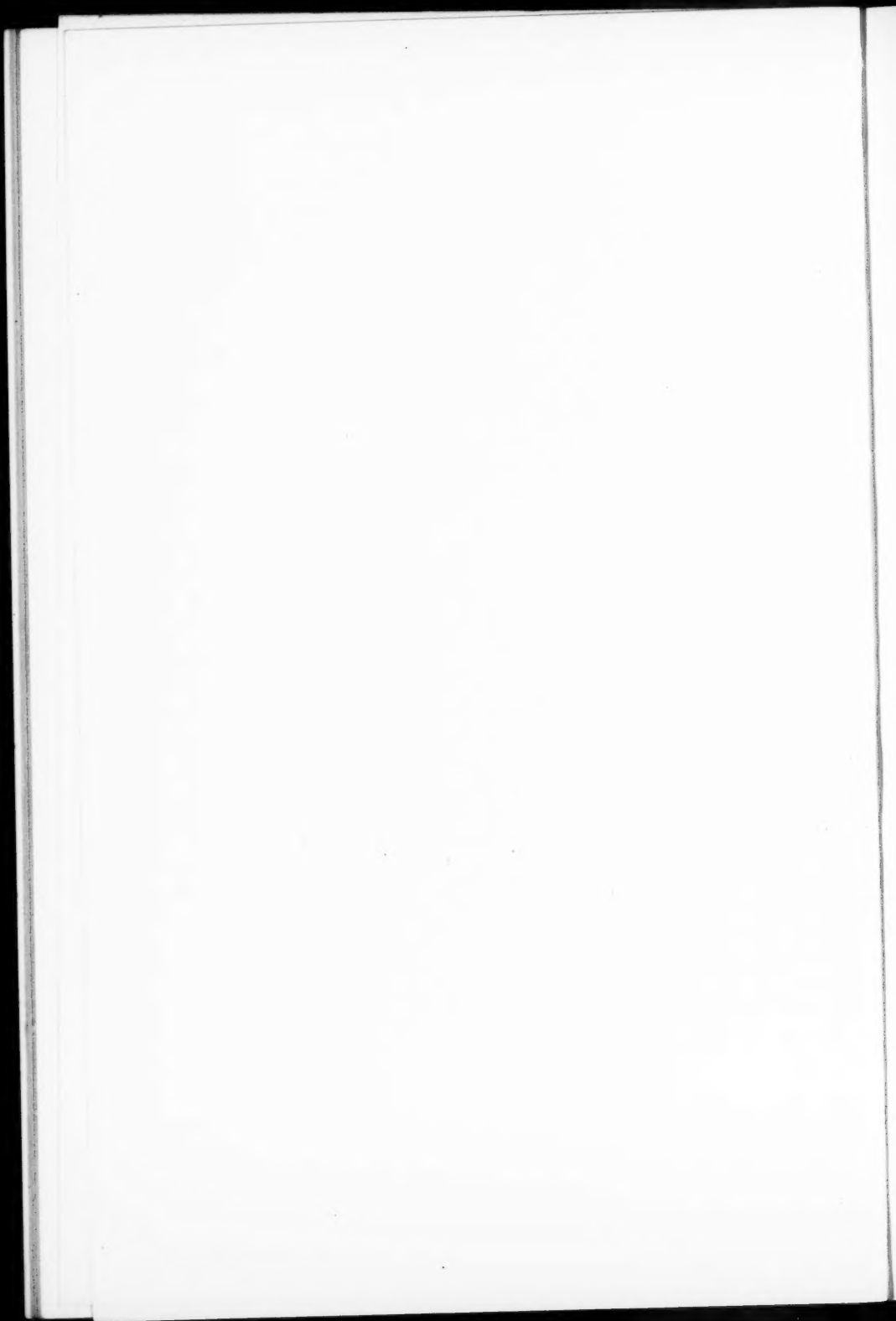


A--Scopometer.



Key to Diagram of Scopometer.

- S. - Source of light.
- Ty. - Tyndall beam.
- Tr. - Transmitted beam.
- Com. - Comparison beam.
- M. - Mirror.
- T. - Target.
- C. - Cell for sample.
- Pl. - Plunger on rack and pinion with scale.
- W. - Wedges.
- P. - Filters.
- L. - Lenses.
- P. - Prism.
- D. - Diaphragms.
- W. - Wicol prisms.
- L-B. - Lummer-Brodhun cube.
- R. - Diffuse reflectors.



adapted to our purposes but also to invent for different needs methods which did not hitherto exist. In short, the work necessitated new tools, and the scopometer represents several such inventions conveniently embodied in a single instrument. It will not be necessary to prolong unduly the present paper with a technical description, because a partial description of the scopometer has been published in the Proceedings of the Optical Society of America and a complete technical description will soon be forthcoming. For the present it will suffice to point out on the diagram that the instrument consists essentially of a tubular optical axis having in order from above downward an eyepiece, a glass plunger which can be raised and lowered by rack and pinion to vary the height of the column of liquid and register this in millimeters on an attached scale (Diagram A). The plunger slides in and out of a glass cylindrical cell which holds the liquid under examination. Beneath the cell is a stage, open in the center to admit light and below this different grooves are provided for the interposition of targets, filters, stops, etc., accordingly as these are to be used for the type of examination in hand. With the illumination, the instrument as described thus far is exactly the same as the turbidimeter I had the privilege of showing you in 1921 for measuring the concentration of albumin in urine. In the scopometer you will note that the same lamp or source of illumination which furnishes the incident light coming from below the cell, also provides a 100 millimeter long Tyndall beam which finds admittance to the cell through an adjustable slot on the front of the instrument. The same source of illumination also provides, when required, a photometric field with light brought through a channel tube which leads to a Lummer-Brodhun cube placed so as also to be in the path of the optical axis containing the plunger and liquid-holding cell. Thus it will be seen that the same source of illumination provides three separate light conditions. Such

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an arrangement is extremely flexible and permits practically any kind or combination of lighting to be brought into play as may be desired for the different purposes for which I designed the scopometer.

It will here be in order to point out that all of the instruments or optical methods which have hitherto been available for biochemical and medical work make use of what is known as the photometric criterion. This is the criterion familiar to biochemists who do their colorimetric work with instruments of the Duboscq type, and may be described as matching the brightness of two juxtaposed separate fields seen with the eye when looking into the instrument. In order to broaden the concept of this criterion so as to have it include every form of comparison I will speak of it as the matching or comparison criterion.

There is another criterion which has not hitherto been available in the laboratory or for medical and biochemical work. It is, I think, the oldest of all optical criteria and was familiar even to the ancients who saw objects disappear into the depths of the sea. It is characterized by the disappearance of an image under observation and has found its greatest usefulness in field work because of its simplicity and freedom from troubles connected with standards. Notwithstanding such advantages, this criterion has never been used in laboratory work such as ours, because the instruments which employ it are impossibly crude. In the case of the scopometer this criterion, which I will call the extinction criterion, has been so refined, and its range of application so extended as to be an important feature of the instrument. From this it will be seen that the design of the scopometer is unique in that it permits the use of both comparison and extinction criteria.

In passing, it may be explained that the extreme development of the extinction criterion attained in the scopometer

was postulated on the several different advantages inherent in the nature of the criterion. Of these, the most striking are: Greater simplicity and handiness, the ease with which the instrument can be kept constant, and the elimination of comparison standards. Others will appear in the course of describing different methods available with the scopometer.

Adapted as it is to research purposes, the scopometer is perhaps too expensive and elaborate for routine work, although its operation is extremely simple and rapid. It was, therefore, decided to incorporate some features of the scopometer in a much less expensive model of the instrument which is extremely handy for routine determinations. This model I call the junior or test-tube scopometer. The operation of this model is so simple, handy and rapid that it is practicable for field as well as laboratory work.

#### COLORIMETRY

As an aid to quantitative chemical analysis, colorimetry is already venerable. The classic treatise on the subject by Kruss dates back over thirty years, and Duboscq made his colorimeter in the middle of the last century. The Duboscq has always been the standard instrument of the chemist who is now fortunately in a position to choose from several models made with a high degree of refinement by different makers here and abroad. In reality, the Duboscq is a color comparator because it compares the color density of a solution of unknown concentration with a known standard solution prepared purposely in the same way, and at the same time, as the unknown for the comparison. When the two solutions are matched in the instrument the depth ratio gives the ratio of the dilutions, and its reciprocal the concentration ratio of the colored components of the two solutions.

Colorimetry has attained its great popularity among chemists because so many reactions give characteristic colors, and

it has proven especially suitable and useful in biochemical and medical work because the phenomena to be measured are so complex, and the specific differences so great, that the correlation of plentiful data is necessary for progress. Furthermore, great sensitiveness is essential because vital substances are necessarily limited in quantity. In addition to these requirements, it is well known that for clinical and routine work speed and simplicity of manipulation are indispensable. This last requirement is emphasized by the somewhat paradoxical fact that although the Duboscq, by constant improvement, has now attained all the perfection of the photometric criterion, there has been a recent unmistakable but decided drift toward simpler or what might be called degenerated techniques.

The turn toward handier but less accurate methods of colorimetry is accounted for by certain features connected with the use of the Duboscq which are regarded as disadvantages of the method, although, of course, every method has its inherent disadvantages. In the case of the Duboscq and other comparison methods the dilution is often so great as not to give sufficiently saturated colors. Again, the method is considered hopeless when the diluent itself is colored. Furthermore, serious questions are involved in the failure of the dilution law itself, this being usually interpreted as involving a transformation in one or more of the colored molecular constituents under observation. Undoubtedly, however, it is chiefly the matter of preparing and manipulating fresh standards that is responsible for the turn toward easier but inferior methods. The very natural desire to economize time, trouble and expense in preparing fresh and generally labile standards for comparison has taken the direction of a demand for permanent standards, which dealers are meeting with colored glasses and more or less permanently colored solutions, generally of inorganic salts. Undoubtedly some of

these are satisfactory when employed under proper conditions and in laboratories which have the facilities and understanding to check up their standards at not too great intervals. But many of the standards which are offered are patently ill-advised and it is apparent that both tendencies, *i. e.*, permanent standards, and comparing colors of test tubes in racks are in the wrong direction because they sacrifice precision on the altar of convenience.

In developing the extinction criterion into satisfactory methods of turbidimetry my experiences demonstrated so conclusively its inherent advantages in such matters as speed, constancy, simplicity and freedom from trouble with comparison standards, that it was not unnatural for me to think of the possibility of bringing these to the aid of colorimetry in order to gain the desired convenience without sacrifice of precision. The results of experiments directed toward this end proved successful far beyond expectations and I was fortunate in being able to develop a simple and rapid method of colorimetry which, in addition to other advantages, completely eliminates the necessity of employing any kind of comparison standard whatever. Only the method applied in the junior or test tube model will be dealt with in the present paper as the smaller model is adequate for all routine insurance work. The larger model allows a choice of several different colorimetric methods.

The method rests on spectroscopic principles, but no spectroscope is employed although most of the experimental work was done with such instruments. For the purpose of understanding the theory underlying the method it will simplify matters to visualize the dispersion of a beam of white light into its visible spectrum, *i. e.*, running from about 4,000 to about 7,000 Angstrom Units or from violet to infra-red running through the blue, green, yellow, orange and red frequencies. The simplest application would be the case of some pure

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color such as the blue copper sulfate solution used in sugar tests which is minus red components. If a layer of this be interposed between the light and the spectrum, an effect is immediately seen in the changes taking place in the red region of the spectrum. Depending on the thickness of the layer used or the density of the blue color, this effect will be more or less in the nature of an obliteration of the red region of the spectrum by a dark band of varying breadth and darkness taking the place of the red color. This is what is called an absorption band and what enables us to say that that blue absorbs red and is therefore complementary to it. If one goes the other way about and examines a red solution that is minus blue components, the same effect is seen to take place in the blue region of the spectrum which we saw take place in the red region. In this case it is said that red absorbs blue and is complementary to it. If we now look through both the red and blue solutions at the same time by superimposing one on the other, it will be seen that by varying the thickness of the solutions we can at will get an absorption band in either the red or the blue region or in both regions, and of any desired intensity.

From this it follows that all of the elements essential for measuring either the red or blue solutions are present, and that by fixing or standardizing one of the colors and varying the thickness of the layer of the other color to a point at which an absorption band of predetermined intensity results, the blue solution can be used to measure the intensity of the red one or vice versa. Experimentally it was determined that the absorption band intensity which is best adapted for the purpose of such measurement is that point at which the extinction of all color takes place. It has also been found that small strips of appropriately dyed gelatin, called color filters, provide a perfect means for fixing or standardizing the conditions of the complementary color. Such color filters



are available in great number and variety, but three of them—a red, blue and green—have been found sufficient for measuring every one of the many colors so far experimented with. It is pertinent at this place to call attention to the fact that in this method it is advantageous to select color filters that exclude all light (*i. e.*, colors) other than that absorbed by the color under examination. In other words, the filter acts to isolate that region of the spectrum in which the absorption band which is to be measured is found.

One would never gather from a description of the method how quickly and simply the actual determination can be made in the instrument. The test tube is placed in such a way that the field diaphragm, color filter wedge and eyepiece for the observer are in front of it, while behind is the target or transparent, short, narrow line of light. If, for instance, one looks through the instrument containing the blue solution, one sees through the field diaphragm the red of the filter. In the middle of this small circle is a short, bright, narrow line. The neutral wedge with a scale is then shifted to a point where the target or bright line disappears or becomes extinct. At this point the scale reading is taken. If one knows the scale reading at which standard solutions such as have heretofore been used disappear in a given test, a very simple calculation gives the desired numerical result. If one is dealing with solutions which are not pure in color but which have several absorption bands, such, for instance, as the product obtained by boiling picric acid solution and sugar, it is just as easy to make the determination as in the case of a simple color. All one has to do is to select the particular absorption band one wants to measure. In fact, this feature presents another distinct advantage of the method in that it is possible to make use of this feature to get rid of interfering colors. For example, the absorption band of phenolsulfonophthalein in the yellow can be used to deter-

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mine its concentration in the urine, notwithstanding the presence of yellow or even bloody constituents because both blood and urine have high transmission in the orange. Again, the hemoglobin of the blood may be measured with a blue filter and its pH with a red filter if a blue indicator such as brom thymol blue be used. In other words, it is possible to measure the concentrations of several constituents of a solution in the presence of each other if regions of the spectrum can be found where the absorption is peculiar to a single substance. To avoid an interfering color, choose its dominant hue and measure the absorption band of that hue.

Another advantage of the method is the very long range which it makes possible. A final advantage of the absorption band method is economy of sample. A 30-mm. depth of fluid may look pale without a filter, while with the filter a 10-mm. layer of the same fluid looks to be quite dense. Finally, it may be said that the Duboscq method becomes more sensitive when such color filters are used. Thus if a color such as the picric acid sugar color is taken as an example, transmitting about 90% in the yellow and 10% in the blue, on doubling the thickness the yellow transmission is reduced to  $.9 \times .9 = 81\%$ , the blue to  $.1 \times .1$  or 1%. If such two thicknesses are compared in the Duboscq colorimeter the contrast ratio in the blue is nine times what it is in the yellow. All of the comparison methods can be improved by using the absorption band principle if the brightness of the source of illumination is increased to meet the increase in density. Of course the extinction criterion is theoretically not as perfect as the comparison or matching criterion because the matching criterion is a so-called null method while the disappearance criterion is largely an absolute measure of visual acuity. Actual experience, however, shows that the sensitiveness gained by using the absorption band principle more than compensates for such differences as there may be between the extinction

and matching criteria. In the scopometer an adaptation field is provided so that when the endpoint is reached the field brightness suffices to comfort the eye and make it more critical. It has been found that the instrument is quite reproducible and that lamps and targets can be replaced without introducing any systematic difference in the readings. Determinations can be made so accurately and rapidly that aside from the expense, there is no reason at all why the smaller scopometer cannot successfully be used by examiners in the field. The instrument, however, soon pays for itself by saving all expense connected with standards.

#### TURBIDIMETRY

The fact that the list of applications for turbidity measurements is extremely long and important, coupled with the fact that even today there is no standard instrument in use in laboratories for making turbidity measurements, both point to the unsatisfactory experiences with such instruments as have been available and their failure to give the desired results. Actually, the scopometer is the result of our failure to get the desired results with different turbidimeters and nephelometers that were tried. It should be noted, however, that when we became interested in the matter, the only instruments available for measuring turbidity were devices employing either freshly prepared or permanent standards for comparison, and they were all comparison methods. After using intensively several different comparison instruments, our experience led me to regard the standard as part of the instrument, and it was to escape troubles with standards and their inconstancies that caused me to try to develop the extinction criterion into a practical method.

In 1921 I had the privilege of showing this society the fourth model of a turbidimeter which enabled us to learn about the nature of suspensions we were dealing with, facts

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that it had not been found possible to learn with then existing instruments. It would be true to say that this instrument initiated us into the difficulties of the problem of measuring turbid solutions and also taught us how little was known about their complexities. Correlation of the results of our turbidimeter with the results of nephelometric and Tyndallmetric measurements yielded more information than either singly, so that this turbidimeter has been further developed into what I now call the scopometer, which gives several different measurements of the same turbidity without taking the sample out of the instrument; in fact, it provides a new tool arranged to correlate with older ones.

When we see a cloudy liquid which we call turbid we are not apt to think how complex it really is even if of the simplest type, or that the turbidity itself is really a composite of a number of different features of the dispersion. The most important of these are: (1) the number of particles; (2) the size of the particles; (3) the shape of the particles; (4) the color of the particles; (5) the difference of index of refraction between the particle, and the liquid holding the particles. With each of these playing its role in the resulting turbidity, it will easily be seen that one type of measurement is particularly sensitive to one feature of the turbidity, while another type of measurement may be sensitive to another feature of the turbidity complex. The scopometric method might be called a covering power method, and seems to give a more general measurement or a measurement less specific to any one feature of the turbidity than any of the methods which have hitherto been employed. The feature of the larger model which distinguishes it from other instruments for the same purpose, is that it allows several different measurements of the same turbidity to be made at practically the same time without taking the sample from the instrument.

Our observations seem to show that the size of the particle

is extremely important in any kind of turbidity measurement. This can be seen very readily by holding two turbidities up to the light and finding their appearance practically the same, such as urine containing protamines and urine containing albumin precipitated with sulfosalicylic acid. The same two turbidities held in the shadow or against a dark background show a great difference between each other, one being scarcely perceptible while the other shows a marked turbidity. Many examples of this misleading difference can be seen and in the few instances in which we have studied them, the difference seems to be due to differences in the sizes of the particles. This is very important because in matching test tubes in a bright light one is not apt to note the difference and will be misled thereby. Undoubtedly this difference also accounts for some of the freak results which have discredited turbidity measurements in general. The scopometer provides the scopometric method, which is a method of covering power and which is, I believe, the first instance in which the extinction criterion has been used in a biochemical laboratory instrument as a criterion for measurement. The scopometer also provides a Tyndallmetric measurement. In other words, the Tyndall beam effect in the liquid under examination is matched against the light from the same source of illumination coming through a permanent arbitrary standard. The photometric measurements seem to be much more sensitive to particle size than the scopometric measurement. In addition to these, the scopometer is provided with a Nicol prism in the path of the light coming through the cell to the eyepiece by which the percentage of polarization or depolarization of the Tyndall effect can be measured.

These three methods have been embodied in the instrument in such a way that they can be carried out in a moment or two and, of course, give much more information about the dispersion to be measured than any instrument providing but

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one criterion of measurement. The whole matter of the optics of turbid media is extremely complex, and while the literature is very extensive there has been a neglect on the part of writers on nephelometry to appreciate or at least to point out that it is probably the technique of reproducing the dispersion rather than deficiencies in the optical instruments which account for the failure of any one turbidity measuring device to come into general use. The scopometer altogether discards freshly prepared comparison standards (although provision can easily be made for using these), not only because of the fact that it is very often impossible to contrive these, but also because unless they are exactly the same and made at precisely the same time as the unknown, the series of changes that take place in both standard and unknown are not the same. The dilution law of the scopometer happens to turn out a simple proportionality which makes calculations simple.

The matter of reproducing dispersions resulting from precipitation is by no means settled except as to certain limits. Certainly the most important matter in the measurement of dispersions at all times is the care with which the technique is devised and carried out with the object of obtaining reproducibility. For most medical work a reproducibility of 10% can be had without great difficulty; in certain instances it has been possible to obtain much better precision. It would be impossible within the limits of a paper such as this to go into the various complexities both physico-chemical and optical which enter into the matter, and some of these will be dealt with specifically and at greater length in forthcoming publications. This is only a general explanation of the working of the scopometer and advantages which it is believed to have over previous instruments. For our albumin work it more than suffices and it is difficult to conceive of any simpler, more accurate, or practical method.

## OTHER MEASUREMENTS

With accessories, other measurements beside color and turbidity can be made with the scopometer. Thus a spectroscopic eyepiece converts it into a very handy and beautiful spectroscope. In conjunction with the Nicol prism, which is a part of the instrument, a target containing another Nicol prism, according to Lippich's principle, converts the scopometer into a polariscope. It is also expected to eventually incorporate in it a new method of interferometry.

## JUNIOR SCOPOMETER

As the scopometer was intended for the study of dispersions and as we became more and more impressed with the complexities involved as we went along with the work, it became necessary to make the instrument more and more elaborate and more and more expensive in every new model, so that it reached a stage where it is probably too expensive and elaborate for routine work. I have, therefore, devised a smaller and inexpensive model which I call the Junior or test-tube scopometer, which works with wedges. In fact, this was the first instrument I designed for measuring turbidity, and I tried to make it about nine years ago but was unable to do so, because at that time no satisfactory turbid wedge was procurable. Last year Dr. P. V. Wells, working in the Prudential Laboratory, happily hit upon the idea of making such a wedge by casting a suspension of fine material in pyroxalin. Dr. Wells is able to reproduce these wedges and at any desired pitch or slope. They are permanent and inexpensive and meet the requirements perfectly. Last year I had the pleasure of showing you the first model of the Junior Scopometer as it was at that time arranged for turbidity measurements, in particular for our albumin test. Since then three instruments have been in use in the Prudential Laboratory for a little over a year now and in the hands of about thirty



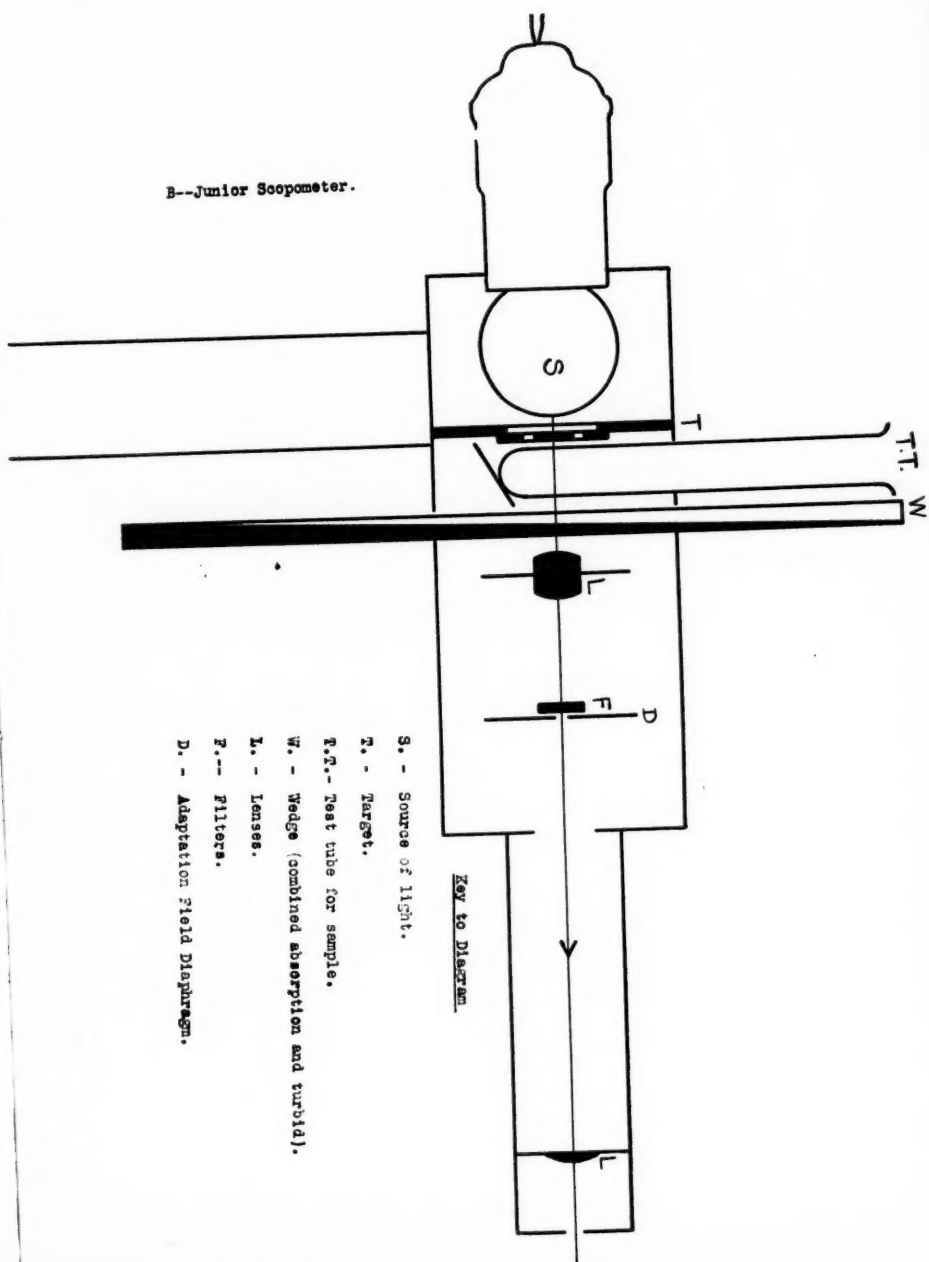
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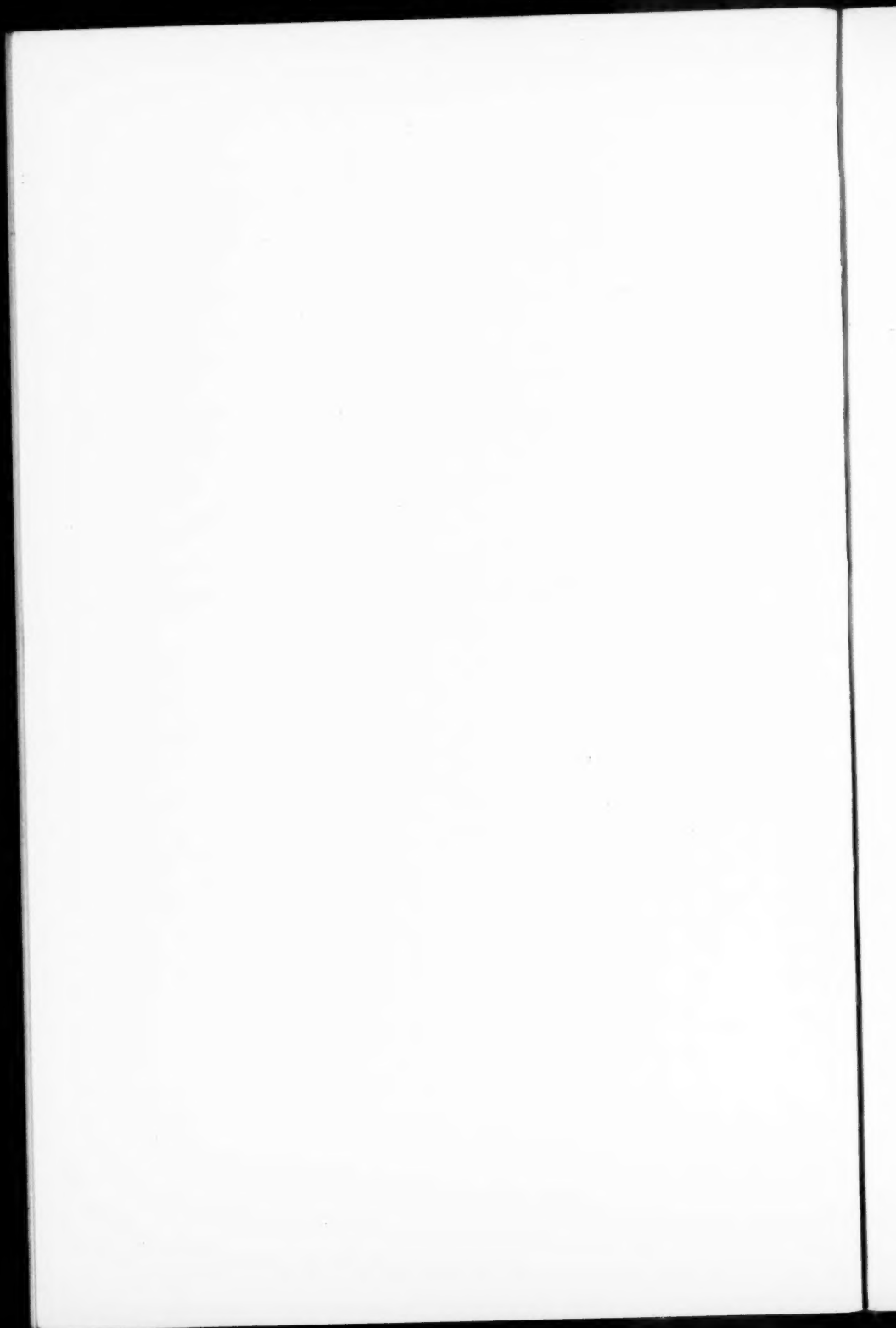
different technicians. Two of the instruments have been used in the routine urinalysis laboratory in making about eighty thousand albumin tests. They have been found perfectly satisfactory in every way, the technicians preferring them to comparing tubes of all kinds, and their use has occasioned no difficulties whatever. We have, therefore, discarded altogether our older series of permanent test-tube standards for making albumin tests. One of the instruments has been employed in connection with experimental work, and during the time Dr. Rose was working on methods for determining albumin and globulin separately. This work involved checking the scopometer results with Kjeldahl nitrogen estimations and gravimetric nitrogen estimations. The checks have been surprisingly accurate. Our experience shows that determinations made with the Junior Scopometer do not take as much time as making comparisons with a series of test tubes, and that the results are much more accurate. It is difficult to conceive of any method simpler than the albumin method we have used during the past year.

Since the instrument was shown the Society, it has been modified only so as to permit incorporation of the colorimetric method described earlier in the paper. It may be said that with this method the results show far greater precision, and with much less trouble, than any arrangement for matching the colors of test tubes in racks. In fact, the precision with the new method in the Junior Scopometer approaches that of the Duboscq under the usual conditions. There are no mechanical parts to get out of order. The wedges and color filters are permanent and the whole design permits such speedy and easy use that an inexperienced person can get good results after two or three trials. In short, the Junior Scopometer is a laboratory instrument which fulfills every requirement as to speed, simplicity, etc., that is needed for field methods. Its cost is soon saved by the elimination of all expense con-



B--Junior Scepometer.





nected with standards, and its usefulness in measuring over long ranges both color and turbidity.

#### METHOD FOR THE STUDY OF ALBUMINURIAS

Supplementing the printed paper, I would like to show you a few slides illustrating types or pictures, as they might be called, of albumin excretion. It will be impossible to more than glance at these at the present time because through the kindness of Professor Sheldon of the Physics Department of New York University and Messrs. Bausch & Lomb it becomes possible for me to demonstrate with a spectrum projector the phenomena underlying the kind of color measurements which are so easily attainable with the junior scopometer. It may be said that the Prudential work on quantitating proteins was instituted in order to obtain such pictures as well as for doing the routine work, because until we were able to make rapid and precise estimations of the amounts of albumin in urine there was practically complete ignorance as to the way in which the different kinds of albuminurics excreted the albumin. It was felt that if we could get this knowledge or information it might lead to better insurance selection by giving the medical director means of knowing definitely the kind of albuminuric he was dealing with in making his selection. These curves or types are made by examining the urine passed during the twenty-four hour period under conditions similar to those of the Mosenthal test. The procedure has been modified in a few unimportant details but we have found it advisable in all such tests to have the two hour specimens passed always before eating meals. In this way we get a more clean-cut picture of the effects of digestion and other factors.

#### DIRECTIONS FOR PROTEINURIA TYPE TEST

Eat and drink what you wish, but be sure to note the time

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when you eat meals (or eat and drink anything between meals). Also be sure to note the amounts and kinds of food and drink taken. It is desirable not to eat or drink anything between meals during the test period.

Pass all urine directly into the clean graduate and measure amount exactly. (The graduate should be cleaned after each measuring by washing out and setting aside upside down to drain.) Then transfer sample to the labelled bottle, discarding what is left in the graduate. Write on the label name, *time* of voiding and the *amount* passed at the time of sample. Keep the samples in a cool place. Proceed as follows, making sure to pass urine always just before eating meals:

About 8:00 A. M. On arising, void urine before eating breakfast; take a sample as directed and note the time of previous voiding.

.. A. M. Eat breakfast. Write down what you ate and drank and note time.

About 10:00 A. M. Void urine; measure and take sample as directed.

About 12:00 M. Void and measure urine and take sample as directed immediately before noon meal, noting time and amount on label and kinds and amounts of food you ate and drank.

About 2:00 P. M. Void urine; measure and take sample as directed, noting time.

About 4:00 P. M. Void urine; measure and take sample as directed, noting time.

About 7:00 P. M. Void urine and measure and note amount and take sample as previously directed before eating evening meal. Be sure to write down what you ate and drank and try not to eat or drink anything more until breakfast next morning.

About 10:00 P. M. Void urine; measure amount and take sample as previously, noting the amount and time on label.

After 10:00 P. M. Measure all urine passed, noting time and amount. Mix this with the urines voided just before breakfast.

About 8:00 A. M. Void urine before breakfast, measuring amount voided since 10 P. M. and taking sample as previously and noting time and amount on label. *Be sure to label properly, especially as to time and amount; cork securely; and write your name on each bottle.*

The specimens are then separately examined and the results plotted. It has been found by experience that the curves tend to fall into about five different groups which have certain general excretory characteristics to identify them. The problem has been on the one hand to classify all known causes of albuminuria and identify them by the type of albuminuria excretion.

#### ALBUMINURIAS CLASSIFIED AS TO CAUSE

1. Orthostatic
2. Adolescent
3. Strains:
  - a. Exertion
  - b. Exposure
  - c. Digestive
4. G. U. infections or inflammations:
  - a. Urethral
  - b. Vesical
  - c. Pelvic
  - d. Renal
5. Haemic:
  - a. Anemias
  - b. Renal irritations secondary to elimination of toxic bodies (*i. e.*, tbc., alcohol, colds, etc.)

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### 6. Cardio-renal:

- a. Decompensation
- b. Congestion
- c. Degenerative
- d. Interstitial nephritis, arterio-sclerosis
- e. Parenchymatous nephritis (secondary and sub-acute)
- f. Nephrosis

The problem has shaped itself to derive sufficient data from rigidly controlled material, and when this knowledge has been gained, to develop further means which would enable a medical director to have at his disposal the information derived from a curve like this with two or three specimens from the individual taken at random times such as it is possible to get in life insurance work. There are many points connected with this future part of the work which it will be impossible to go into now.

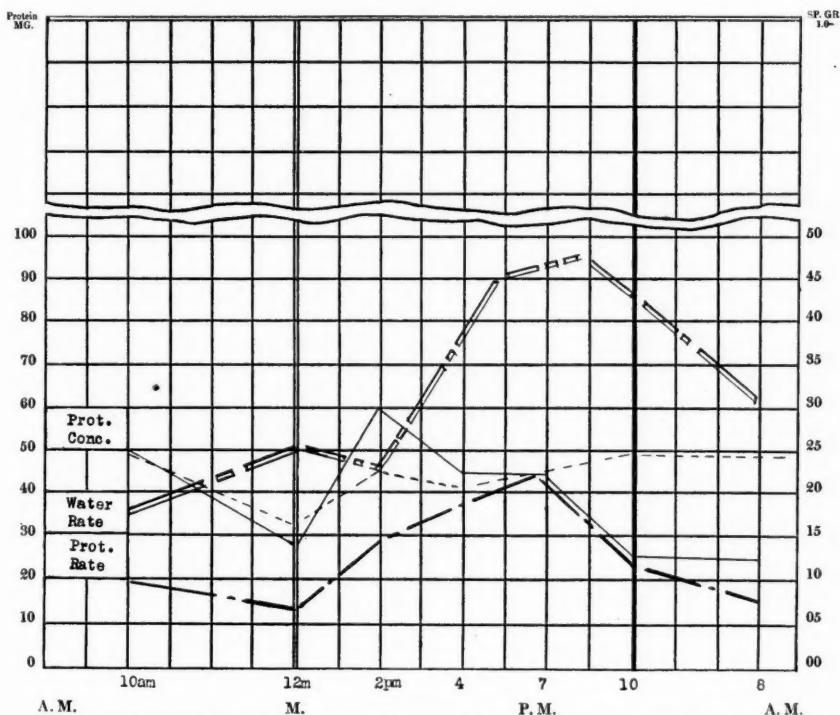
The first chart shows the manner of excreting protein of a man 49 years old who has been under my personal observation for more than thirty years, during which time his urine has never failed to show a well marked ring by Heller's test. He has been singularly free from symptoms and very active in business and swims, hunts and golfs. Recently he was operated on for an old hernia after a most thorough examination including cardiogram, basal metabolism, blood chemistry, dye tests, etc., all of which turned out to be negative. Note that at 2 P. M. the tested urine shows 60 mg. while at 8 A. M. the tested urine shows only 22 mg., or one-third as much as it did at 2 P. M. Note, however, that at 2 P. M. he is excreting 54 mg. as against 150 mg. at 8 A. M. In other words, the urine test shows more albumin at times when an individual may be excreting less albumin. This means that the results of albumin tests will be often misleading unless

CHART 1.

## PROTEINURIA TYPE

Name..... P. E. ....

Clinical Diagnosis.....



KEY	Urine, Excretion	_____	Protein, Concentration	_____
	Rate	_____	Excretion	_____
	Sp. Gravity	_____	Rate	_____
	Albumin Excretion	_____ x _____ x _____	Globulin Excretion	_____ o _____ o _____

Time:		Date						
		10am	12m	2pm	4	7	10	8am
DATA	Urine vol., cc.	70	100	90	160	280	250	600
	gravity, 1.0-	25	16	22	20	22	25	22
	Protein conc., mg. per 100 cc.	50	28	60	45	45	25	25
	Protein excretion, mg.	35	28	54	72	126	63	150
	Protein rate, mg. per hour	18	14	27	36	43	21	15
Globulin excreted								

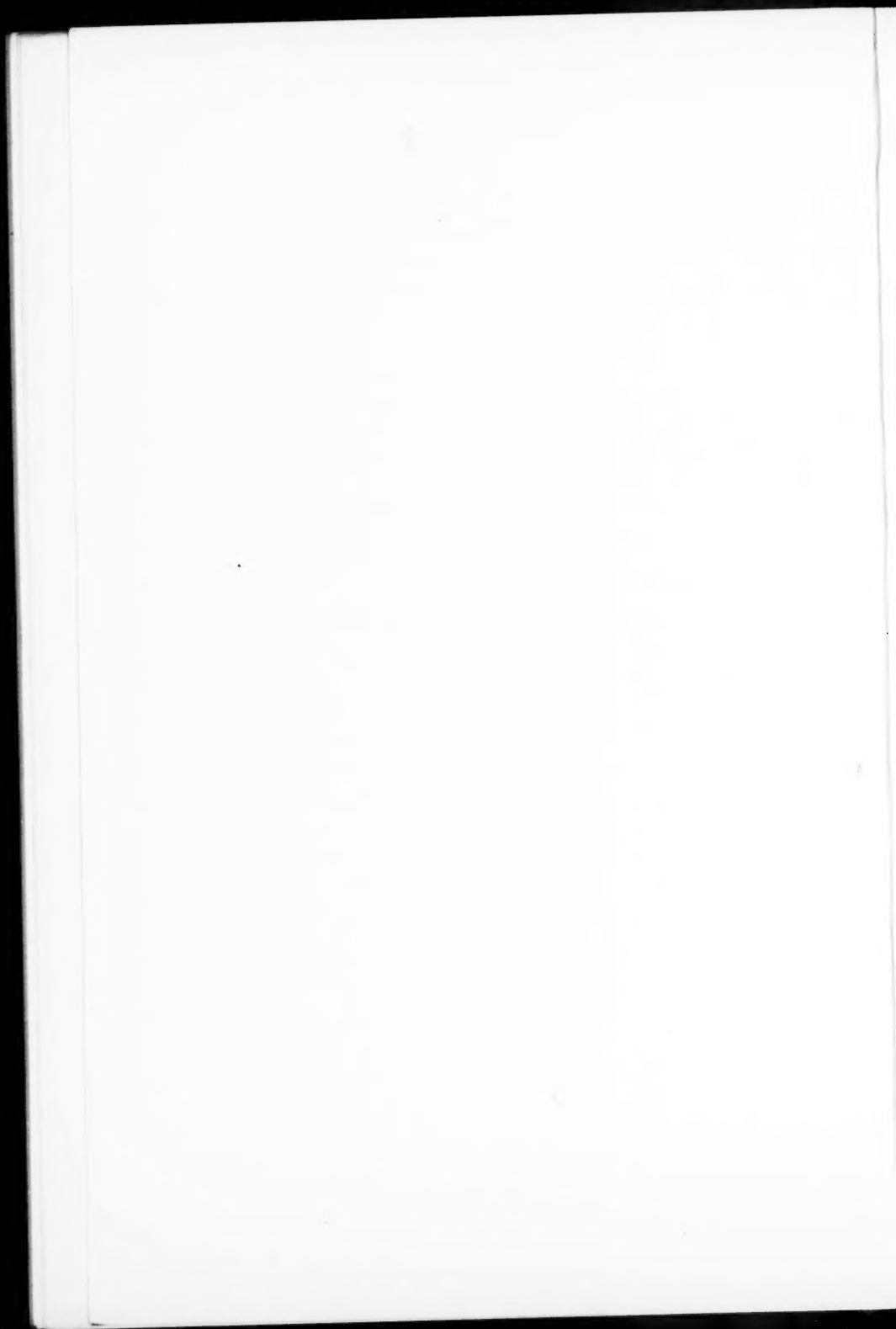
Meals at  $\Delta$ : A. M.

P. M.

P. M.

Fluid taken at  $\perp$ :

(See also other side)



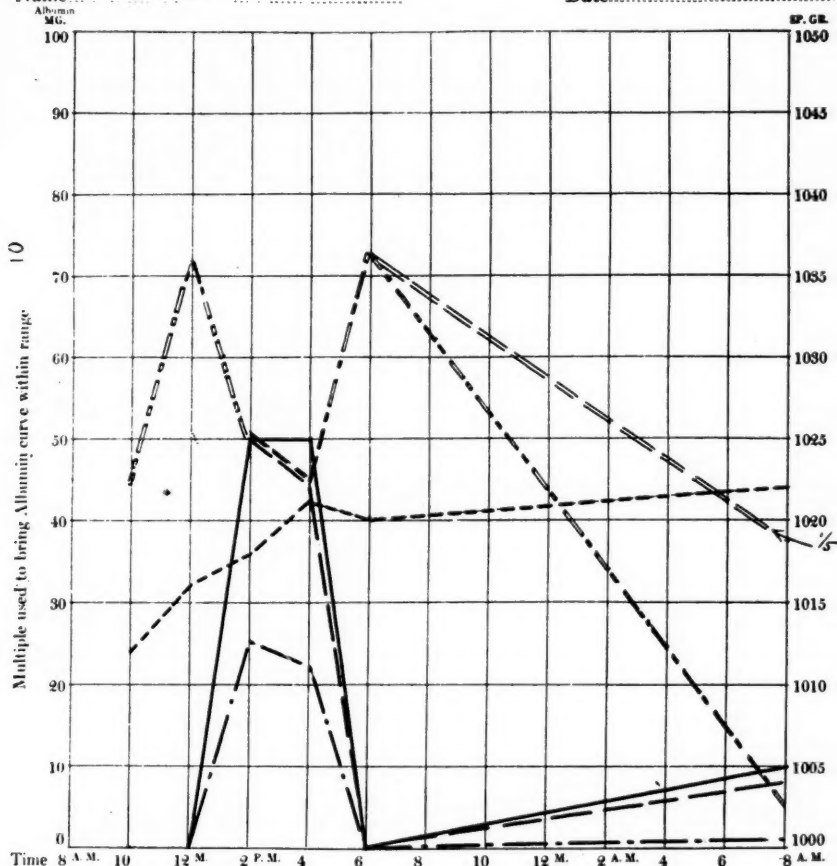


# CHART 2.

## ALBUMINURIA TYPE

Name Roosevelt (3)

Date \_\_\_\_\_



Food and drink						
Vol.	92	145	100	55	145	75 C. C.
Sp. Gr. 10.	12	16	16	21	20	22
Alb. mg.	0	0	50	50	0	1 Per 100 C. C.

KEY

Albumin Concentration . . . \_\_\_\_\_  
 Total Albumin Excretion . . . \_\_\_\_\_  
 Rate of Albumin Excretion . . . \_\_\_\_\_  
 Specific Gravity . . . . . \_\_\_\_\_

SEE REVERSE  
SIDE FOR  
OTHER DATA

44821

FOOD, O. DRINK, +. FOOD AND DRINK, O.

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Urine Excretion \_\_\_\_\_  
 Rate of Urine Excretion \_\_\_\_\_



the volume of urine or, as a substitute, perhaps the specific gravity be taken into consideration. Almost every chart shows one or more such instances, and it is a practical point that is overlooked when random specimens are examined and appraised.

Note also the effects of taking food by the rise in albumin concentration after meals. This characterizes many cases of certain and generally benign types, although the effect is sometimes mixed with that of muscular effort. Note that in this case there is no after dinner rise. In fact, the concentration remains on a level for some time and then falls to the sleeping level. This test was made during the course of an active day's work which ceased just before dinner, and it is probable that the effects of food ingestion are balanced by the effects of rest in this case. Note that the total excretion has no tendency to be flat or of the order of a straight line; also that there is a marked peak running upward from the base line of the curve. This is a type in which the protein excretion seems to go up with muscular exertion and digestion and to go down with rest. The specific gravity is variable and there is no tendency to parallelism of any of the curves.

The second chart is shown merely as an example of one of the several different types of intermittent albuminuria that have come to our notice. I wish it were possible to go into more detail regarding these cases as they are the kind that we are specially apt to meet with in insurance work. Note that in this case the protein goes up to 50 mg. only between noon and 6 o'clock, also that there is an unusual tendency for the protein to go up during the night.

We recently had a particularly interesting case of intermittent albuminuria. It was the first one of its kind I have been able to run down, but surely others are met with from time to time in insurance work. A man applied for the

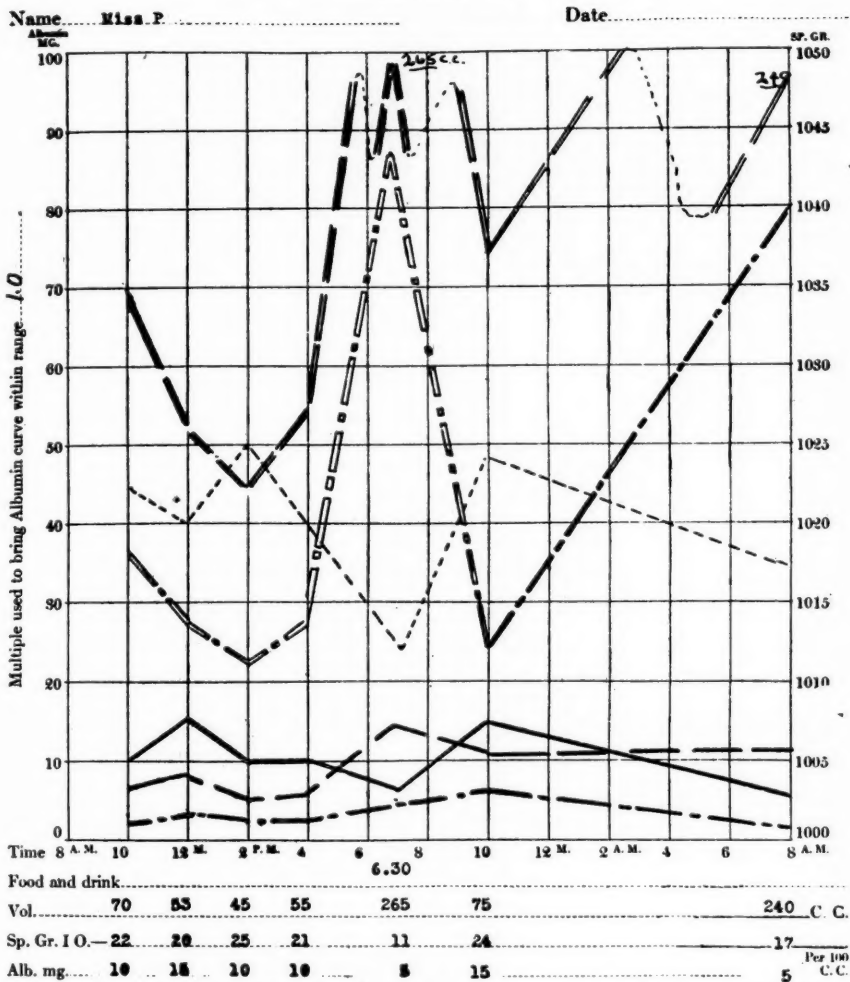
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Longevity Service and his urine was found to contain albumin. When this was brought to his attention he stated that he had been rejected for albuminuria and it was not surprising to him. It was suggested that a 24-hour test be made and the result of the test showed that he had from 40 to 60 mg. of albumin in the urine between the hours of 2 and 4 o'clock. All of the other samples were free of albumin. It turned out that this man was a druggist in a southern town and the early afternoon being his least busy time he was always examined for insurance between 2 and 3 o'clock. He had the habit of going home for dinner on his bicycle about a mile away and doing chores about the house and then riding back to his store on the bicycle.

The third chart shows the case of a lady who took a periodical examination that was offered by her employer, although she stated that she felt perfectly well with perhaps a tendency to be nervous at times. You will note that the albuminuria type is entirely different from both of the preceding. Her blood pressure on examination turned out to be 200. Note how the specific gravity goes up and down with the water excretion, and also note that although the amounts of albumin are very slight the rate is constant through the twenty-four hours as represented by the broken line at the bottom of the chart. This type illustrates the parallelism of some curves and the constant rate of albumin excretion which is not influenced at all by the water excretion. Although by no means final, other curves which we have examined show the effects strikingly and we have come to think of such curves as indicating interstitial nephritic or arterio-sclerotic conditions. In this case death occurred within six months from uremia. Please note the very small amounts of albumin as contrasted with the extraordinarily large amounts found in much more benign cases.

# CHART 3.

## ALBUMINURIA TYPE



KEY

Albumin Concentration . . . \_\_\_\_\_  
 Total Albumin Excretion . . . \_\_\_\_\_  
 Rate of Albumin Excretion . . . \_\_\_\_\_  
 Specific Gravity . . . \_\_\_\_\_

SEE REVERSE  
 SIDE FOR  
 OTHER DATA

44821

FOOD, O. DRINK, +, FOOD AND DRINK, @.

PRINTED IN U. S. A. by Prudential Press

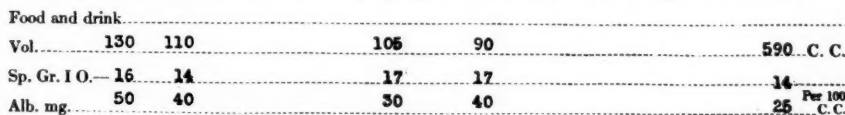
Urine Excretion

Rate of Urine Excretion



ALBUMINURIA TYPE

Date May. 1925.



Albumin Concentration . . .	_____
Total Albumin Excretion . .	_____
Rate of Albumin Excretion .	_____
Specific Gravity . . . . .	_____

SEE REVERSE  
SIDE FOR  
OTHER DATA

44821

FOOD, 0. DRINK, +. FOOD AND DRINK, 0.

Printed in U. S. A. by Presidential Press

### Urine Excretion.

Rate of Urine Excretion,            g. /            hr.



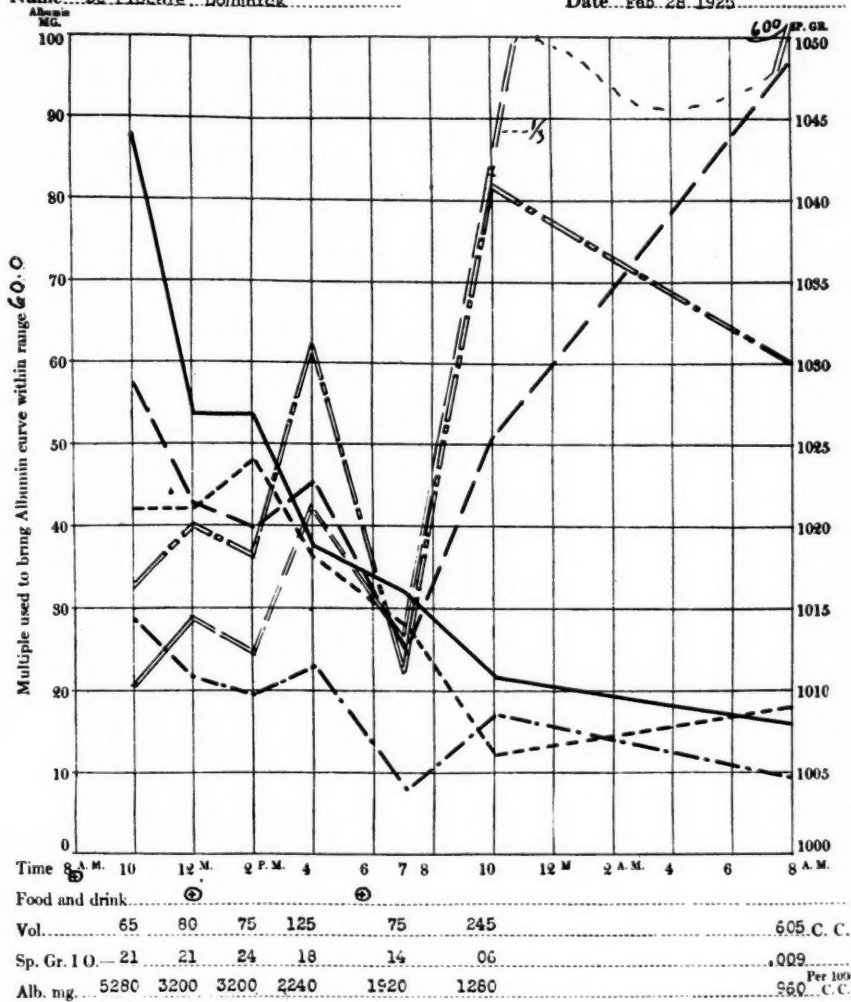


CHART 5.

## ALBUMINURIA TYPE

Name... De Flochie, Dominick.....

Date... Feb. 28, 1925.....



KEY

Albumin Concentration . . . \_\_\_\_\_

Total Albumin Excretion . . . \_\_\_\_\_

Rate of Albumin Excretion . . . \_\_\_\_\_

Specific Gravity . . . . . \_\_\_\_\_

SEE REVERSE  
SIDE FOR  
OTHER DATA

44821

FOOD, C. DRINK, +. FOOD AND DRINK, S.

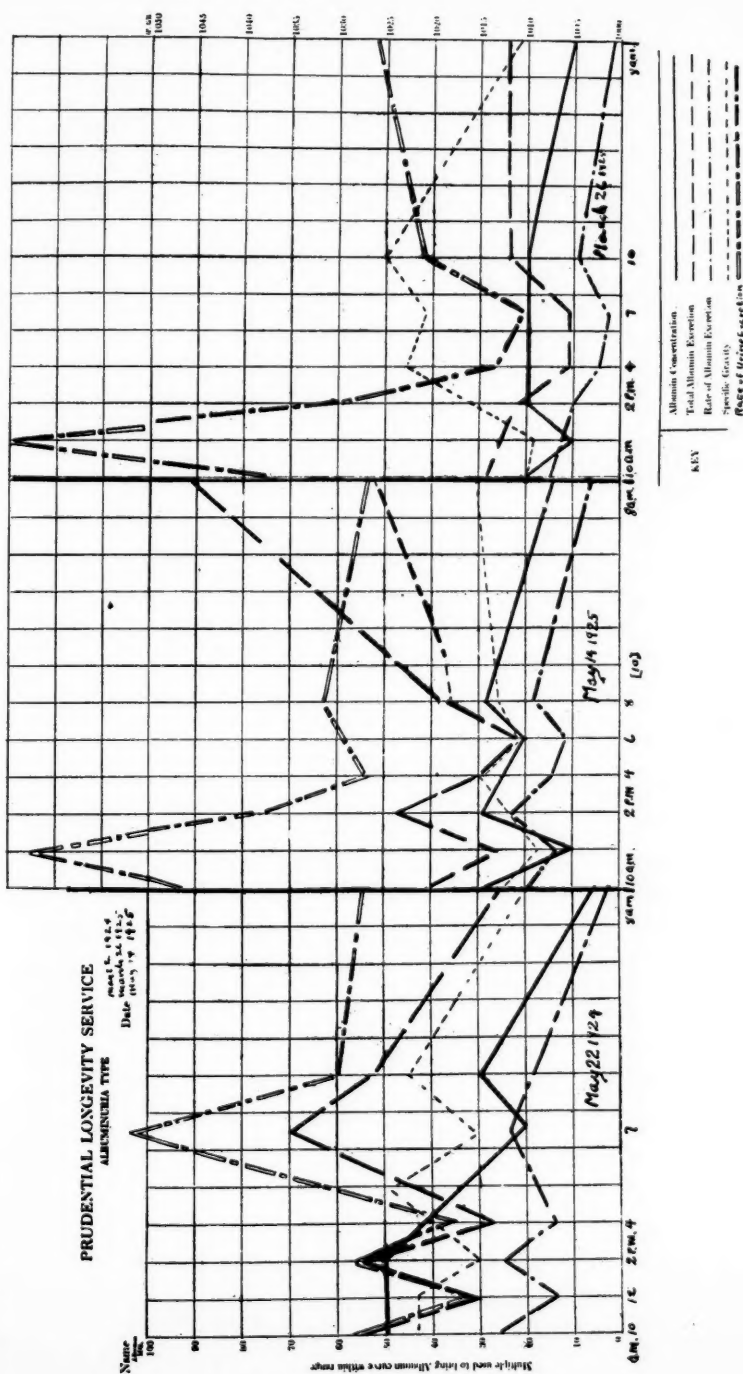
PRINTED IN U. S. A. by Prudential Press

Urine Excretion

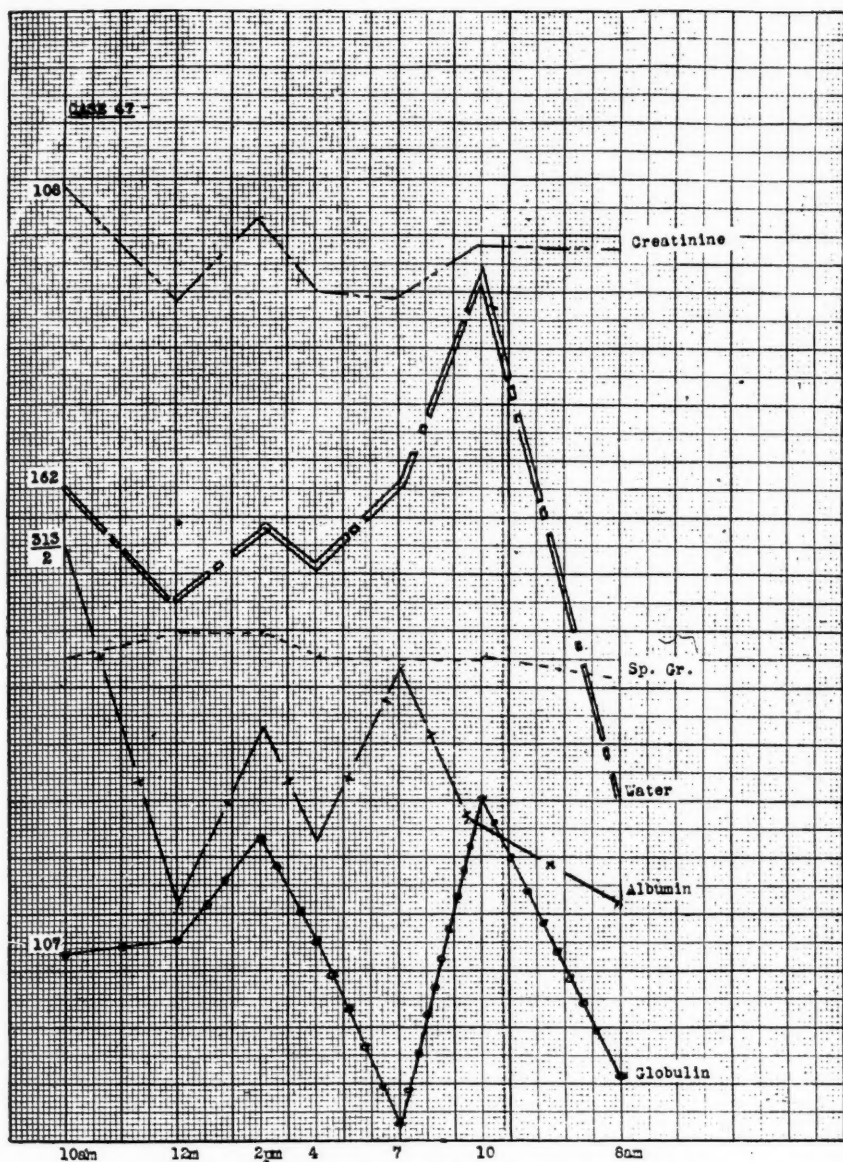
Rate of Urine Excretion — — — — —



CHART 6.

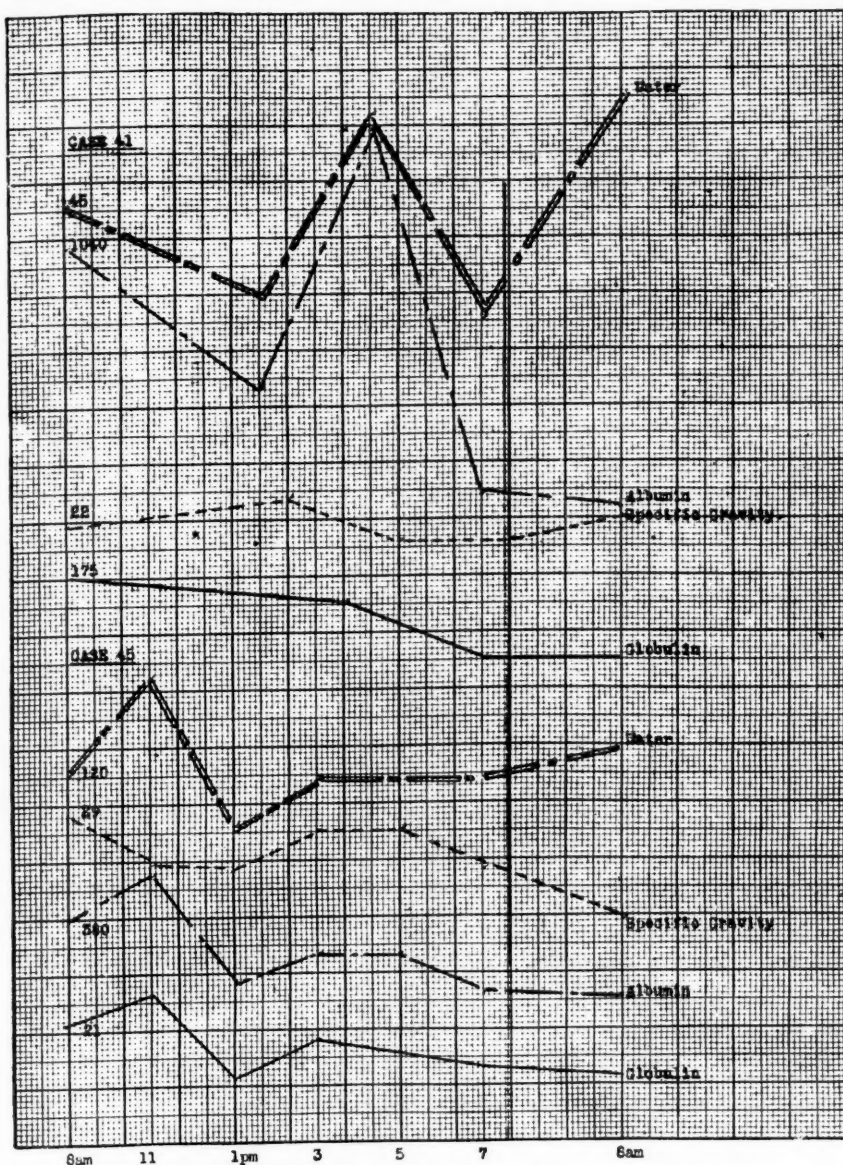






Case 47. Curves from Prudential Proteauria Type Chart spread to show relation between albumin and globulin excretion rates.





Cases 41 and 45. Curves from Proteinuria Charts spread to show differences in relations between albumin and globulin excretion rates.





The fourth chart is shown merely to illustrate the parallelism between the water curve and the rate of albumin excretion. This accounts for the fact that low gravity urine in certain types of cases will show none at all or the most minute amounts of albumin when specimens of good gravity had from the same case will show large amounts of albumin. In other words, it illustrates the dilution effect on protein, and may easily cause one to mistakenly regard a persistent albuminuria as an intermittent one if a delicate test be not used.

The fifth chart, you will note, is different from the others and represents a very interesting type, a sort of staircase effect. The albumin is highest in the morning and keeps going down all during the day. Note the very large amounts of albumin as the multiple of 60 has been used to bring the curve within the range of the chart, in the morning being as high as 5280 mg. per 100 c.c. Note that the volume of urine excreted does not seem to affect the albumin excretion, quite a difference from the case I showed you where these were parallel.

The sixth chart is interesting because it shows several tests made on the same individual over a year apart. We have since made another test of this individual and it shows that cases, at least like his, run true to type over long periods of time. The later charts were made after we changed the method of collecting specimens so as to make more clean-cut the effects of digestion.

There are many points in connection with these charts which are of great interest, but from what has been presented it will be seen that the proteins are excreted differently in different kinds of cases and that by close study of the relationships existing, for instance, between albumin and globulin and creatine and water and other features it may be pos-

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sible by doing several simple tests in the laboratory to decide just what type of albuminuria one is dealing with even with the random specimens obtainable in life insurance work.

Dr. Hobbs—Dr. Folin, may we have a word from you now, sir.

Dr. Folin—I should like to say that I am very glad to note that there is not much need for saying much of anything, for it seems to me that the actual workers in the field are now covering the subject in the right way and in the right spirit, and when we hear these different men come and express different opinions, we know that they are not asleep, and, after all, Dr. Benedict and myself feel that we should come here and see that they aren't asleep.

It is, to my mind, very nice to know the implication of the discussions and practices that are now in vogue. We don't talk any more about urines which have no sugar and about others which do have it. We are talking now in a quantitative manner. That aspect alone, I think, will, in the course of time, bring about a more correct attitude and will bring with it an idea as to how to rate sugar in the urine. Those of us who have been working in that field for many years still aren't quite agreed as to what that sugar is. We determined the quantities and formulated different opinions as to how much of it is glucose, and glucose is the only sugar, of course, that you are interested in, but since you cannot determine glucose separately from the other sugar, you determine the whole and you base your opinions on the total.

Now Dr. Clark still supplements his quantitative methods by the qualitative tests. I would suggest that the development ought to come in doubtful cases. It isn't that we are going to check up the quantitative sugar test with a qualitative one, but we are going to check it up with a different quantitative test. For example, when you are attempting the picric acid test and are in doubt and use some form of copper test, make

it a quantitative one so that you are all the time sticking by the quantitative aspects, for it is through those aspects only that you can enumerate statistics that will stand the test of time.

Also there is just one word in passing that I want to say. I am not afraid now that you are remaining stagnant, but there are great difficulties in promoting scientific work in connection with insurance problems. Just because you have so many tests to make and so many records behind you, it is very difficult to introduce something new. You ought not to introduce something new unless you have extremely good reasons for it. In the case of the transition from the qualitative tests to the quantitative ones, it was comparatively safe and easy to make such a proposition because it was relatively so certain that any fair-minded person could be convinced that the quantitative work was the correct one to pursue, but it is different when you come to change from one quantitative process to another. Then you have to go slow and that is in a way one of the handicaps for progress. You can not promptly adopt new technique because it is better than what you had before, and the only solution for that inherent difficulty, as I see it, is that you all the time carry on more or less side work, check up the tests by new ones, just as Dr. Clark is doing with the old qualitative ones, by parallel processes, but don't let it interfere with your current statistics until you have a mass of material showing where you stand so that when you do want to change, you can connect the new with the old.

Dr. Hobbs—I will call upon Dr. Benedict for a few remarks.

Dr. Benedict—Mr. President: I have very little to add. As Dr. Folin mentioned, it is very satisfying to sit back and see the Companies work out the practical sides of these problems. It is quite impossible for us working in a laboratory

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in comparative isolation to encounter the types and varieties of materials that you meet in insurance work, and while we attempted to cover as many samples as we could, we could not meet the exceptional and peculiar samples that come in to you in relatively large numbers because of the enormous volume of material which you handle.

Dr. Clark was almost apologetic and excused himself for using one or two processes in addition to the ones we have suggested. I think that it should be just the other way, that checking up these processes in every possible way is the ideal thing to do. We know what may be expected theoretically, but when it comes to the insurance laboratory, it may be a very different proposition.

I would say that the introductory words of Dr. Muhlberg's article, in which he states that as many as 50 per cent of the companies have already gone over to at least one of the quantitative methods, is to my mind almost startling. I had supposed that the mass of statistics was so great back of the insurance business that the companies would be exceedingly slow in shifting over, and I think it can be regarded as very encouraging that as many as 50 per cent have already adopted these methods and that a majority has in mind adopting it as soon as possible. I thank you.

Dr. Hobbs—Are there any questions or further remarks in regard to the subject now under discussion.

I want to say that we all are very much indebted to Dr. Muhlberg for his paper.

We will now have an exhibition by Dr. Exton of his scopometer.

Dr. Exton—We have slides showing studies Dr. Wells made on a man at intervals of a year. We made one last week which covers a period of two years, and they are all practically the same. This simply shows results obtained with the instrument as compared with the theoretical, as we

call it, dilution curves of a copper solution and using a red filter and on the Benedict picric acid test using a blue filter.

This shows a study made of the Benedict picric acid sugar reagent with three different filters which increase the range. In other words, at the present time you use five tubes and get a factor of five in your range from one-tenth to five-tenths per cent. By the use of this filter, you get a range of 500, that is from one-tenth to about ten per cent, which would save the necessity of repeating the test when you ran over the one-half per cent tube. These curves also show successive readings, showing the time changes or the fading in the tubes.

Dr. Hobbs—Are there any remarks to be made or questions to be asked Dr. Exton? Does anybody want to speak about anything that has been brought out in his demonstration?

Dr. Kingsbury—I would like to ask Dr. Exton if he finds the so-called Mosenthal effect with albumin when the albumin rate or the amount of albumin is low. Suppose the specimen runs around 10 milligrams of albumin per 100 c.c., is there any Moesnthal effect there?

Dr. Exton—In two of those slides, one showed where there was a case of great albuminuria. I simply say about that, that that case is different. You do get cases of low grade albuminuria who do give the Mosenthal effect, except in the very extreme cases where the rate of excretion seems to be absolutely constant such as the case of this woman. She had, as I remember, 10 or 15 milligrams of albumin, although her kidneys seemed to be working all right.

Dr. Kingsbury—It has been shown before with large amounts that you have that Mosenthal effect, but it seems peculiar that you should have a fixation effect with a substance which is not a normal excretory substance at all.

Dr. Hobbs—Any further remarks or any further questions? If not, this session will stand adjourned until the dinner at 7:00 o'clock.

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### SECOND DAY

President Hobbs in the chair. The meeting was called to order at 9:30 o'clock.

The Secretary announced that he had cast a ballot as instructed for the election of the officers and members of the Executive Council placed in nomination on the preceeding day, as follows:

President,	Dr. Wesley W. Beckett
1st Vice President,	Dr. Robert M. Daley
2nd Vice President,	Dr. J. Allen Patton
Secretary,	Dr. Chester T. Brown
Treasurer,	Dr. Charles L. Christiernin
Editor of the Proceedings,	Dr. Eugene F. Russell

#### Members of the Executive Council:

Dr. G. A. Van Wagenen  
Dr. E. W. Dwight  
Dr. Wm. Muhlberg  
Dr. Ross Huston  
Dr. Morton Snow

These officers and members of the Executive Council were declared duly elected.

Dr. Hobbs—We now have the privilege of having Dr. Mathews with us. The subject of the gall bladder is one which has interested us in the New York Life very much, and I am quite sure it will be an interesting topic for you. The paper of Dr. Mathews is in your hands. It was on the table yesterday and is on the table today. Dr. Mathews said that he would follow the custom of this Association and have his paper published in the same way that we have our papers published, so that the discussions may take place afterwards. Dr. Mathews, may we hear from you now? I have known Dr. Mathews for a great many years. He is one of our leading surgeons in New York City.

OUTLOOK FOR LIFE AND HEALTH OF THE GALL  
BLADDER PATIENT

By FRANK S. MATHEWS, M. D.

*New York*

This paper has been prepared with the co-operation of the New York Life Insurance Company. In fact it was Dr. Angier B. Hobbs, of that company, who suggested that they would be willing to undertake an investigation of cases of cholecystectomy with the idea of determining their life expectancy, a matter quite different from that of surgical mortality. With this investigation in view, I have turned over to the New York Life Insurance Company my operative records of 350 consecutive cholecystectomies and they have endeavored to trace the patients to determine whether they are living or dead and compare their outlook with that of the general population of similar age. But before proceeding to this part of the paper, it may be of interest to your society to present some of the modern physiological and pathological views regarding the gall bladder and its inflammations.

The liver, the largest viscus in the body, has been found to possess a considerable number of functions and probably no physiologist would be willing to assert that our knowledge of the list of functions is complete. The study of its functions has always seemed difficult, the impairment of one function not necessarily coinciding with an impairment of the other functions. Hence no test has been devised which will give us an idea of the capacity to do work of any particular liver for the tests so far devised test one function only. A list of its functions would include the glycogenic function, proteid metabolism with the formation of urea, the detoxication of poisonous substances and the elimination of abnormal substances from the blood, the formation of fibrinogen, secretion of bile and the elimination of bacteria.

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Bile has always been thought to be both a secretion and an excretion. The belief in its secretory character is based on the fact of its partial reabsorption into the blood, on the fact of its duct entering the intestinal tract so high up and the fact that fats are somewhat less well digested when bile does not enter the intestine. However, we must admit that one may live for years in comparatively good health while all his bile is being discharged upon the body surface.

From the surgeon's standpoint, the bile may be considered as a fluid which contains three important materials in solution and in the order of diminishing abundance they are bile salts, bile pigment and cholesterin. The origin of these substances has been the subject of much study. There seems to be little question that the bile salts originate in the liver itself as a product of the metabolism of the liver cells. When the bile is discharged into the intestine the salts are known to be reabsorbed. This cannot be considered a complete absorption. If it were, while the liver is continually forming bile salts and they were continually being reabsorbed, there would arise an unlimited accumulation of bile salts in the blood. It is an accepted belief that bile pigment represents the breaking down of hemoglobin. In this disintegration of the hemoglobin molecule about 5% by weight is separated as a pigment containing iron and from this the iron is set free leaving an iron free pigment known as bilirubin. There has been much discussion regarding the location of the formation of bile pigment. Is it in the liver alone? Is it in selective tissues or more or less in all tissues? A contusion with a black and blue spot represents hemoglobin broken down in connective tissue. Probably red blood cells may break down in any part of the circulation but the liver, spleen and bone marrow are considered the most important locations of destruction of red blood cells and hence liberators of pigment. The type of tissue in which this occurs is commonly spoken of as



the reticulo-endothelial system. The cells of this system in the liver are known as the Kupffer cells and are considered to be endothelial in type and connected with the venous blood vessels. All the cells of the reticulo-endothelial system are definitely phagocytic.

Cholesterin is found in the blood in small amounts and in small but variable amounts in the bile. According to an older theory cholesterin was not considered a true constituent of the bile but was thought to be a product of the lining epithelium of the bile passages and was increased by any catarrhal processes which affected them.

A great deal of experimental work has been done on animals in recent years with the object of determining the functions of the gall bladder. To this knowledge obtained by experiment, the work of surgeons, Roentgenologists and investigators with the duodenal tube in man have contributed. The gall bladder is ordinarily said to have a capacity of 50 cc. but it can dilate to a very much greater size, even several times what we call normal, without rupture. A gall bladder is present in some species of animals and absent in some closely related ones; and this has been thought to indicate that the organ is without function. Though this deduction is not believed correct, yet we must admit that it may be removed in man without any evidence of impairment of body function under the ordinary conditions of life. In the patients without gall bladders the common duct regularly dilates and presumably the sphincter of Oddi maintains a lesser tone. At any rate in animals without a gall bladder, its tone is said to be considerably less than in those with one. The secretion of bile is continuous but variable. The amount secreted at night and during starvation is much less than during the periods of active digestion. A comparison of the bile of the hepatic duct and that of the gall bladder shows a great difference in the degree of concentration, and it is now considered that the

chief function of the gall bladder is that of concentrating the bile as it is secreted by the liver to a volume of one-eighth to one-tenth as much. Under usual conditions, according to Aschoff (1), the absorbed constituents are chiefly water and inorganic salts but under unusual conditions as, for instance, when a stone blocks the cystic duct, the gall bladder may become distended with mucus with entire disappearance of bile salts and pigment. When the colored bile is replaced by the colorless mucus, we speak of the condition as hydrops of the gall bladder. When a similar replacement occurs in the common duct, we speak of "white bile."

As regards the other function of the gall bladder, that of storage of bile to be released when needed, there is very considerable difference of opinion. As generally stated, bile accumulates in the gall bladder during the intervals of digestion and is discharged when food enters the duodenum. The discharge, however, would seem not to be complete for one finds the gall bladder both in man and animals, living or dead, to contain some bile except in the seriously diseased ones. Indeed, as a rule at operation, it is quite full and tense as we would expect since operations are usually performed after twelve to eighteen hours of starvation. Meltzer (2) some years ago announced his theory "of contrary enervation" of the gall bladder and believed that a stimulus applied to the duodenal mucosa produced reflexly at the same time a stimulus to the musculature of the gall bladder and an inhibition of the sphincter of Oddi. This theory, in quite recent times, has been called in question by Winkelstein and Aschner (3) and by Graham (4). Graham thinks the layer of muscle in the gall bladder wall simply gives it a constant tone and says that no one has been able to show contraction of the gall bladder in the open abdomen visible to the eye in response to either mechanical, thermal or electrical stimulation, a type of stimuli which would readily produce a peristaltic wave

in the stomach or intestines. On the other hand, when the duodenum is open, bile has been seen to enter the duodenum in jets from the papilla. According to Graham, then, the emptying is due to relaxation of the outlet rather than to any increased contraction of the gall bladder itself. He thinks, moreover, and so does Carlson (13) that the closure of the duct is more due to the tone of the duodenal wall through which the duct passes than to any definite sphincter of Oddi. Carlson (13), from observation on animals, is not impressed with the intermittent flow. He says that in experimental animals the gall bladder is found in about the same condition of distention whether they are digesting or not. He says that in starvation, even when long continued, in man and animals, that bile is being discharged into the duodenum. Winkelstein and Aschner (3) think that the relaxation of the duct which permits the entrance of the bile into the duodenum is never a complete relaxation but just a partial reduction of tone. In the open abdomen, we expect to find bile held in the gall bladder under considerable tension. Firm pressure is necessary to empty it and experimentally this has been found to amount to something less than 150 mm. of water pressure. However, we explain it, there is no question that bile is continuously secreted in variable quantity and that it is not being discharged into the duodenum at the same rate with which it is secreted. The experiments with the duodenal tube and magnesium sulphate application and the cholecystogram, are conclusive as evidence that bile can be discharged intermittently. Pigments and insoluble powders have been injected into the gall bladder and have been found not to be completely eliminated for one to two days. Bile, as secreted by the liver, is modified by the addition of a considerable quantity of mucus which is added in its course through the ducts and gall bladder. Howell (5) says that this substance is not a true mucus. The gall bladder itself shows no definite mucus glands but they

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are seen in fair number in the ducts. However, after closure of the cystic duct, the gall bladder distends with mucus which it would seem must be derived from the epithelium of the gall bladder wall.

There is a difference of opinion as to the mechanism of the absorption of bile in jaundice. The question is whether it takes place through the liver cells and directly into the capillaries or through the lymphatics and thus into the thoracic duct.

The problem of gall bladder functions under normal human conditions has been considerably clarified by the aid of the X-ray and an ingested dye. Rowntree experimented with tetrachlorophenolphthalein with the purpose of testing liver function and found that this substance is not eliminated through the kidney but is secreted by the liver in the bile. Graham and his associates, in the last few years, have developed a technique using similar compounds but replacing the chlorine atoms by the heavier atoms of bromine or iodine (tetraiodophenolphthalein). When these substances are in the bile, they cast a definite X-ray shadow. As clinically employed, the dye is administered either intravenously or in specially prepared pills at night and on the following morning, the patient taking no food, a series of radiograms is made. In the normal person one expects to see the gall bladder clearly visualized on the plates. Later in the day food and especially fatty food is taken and very shortly thereafter another radiogram is made, when one will find that the gall bladder shadow has almost or quite disappeared. The test is proving of value both as to our knowledge of normal physiology and also of pathology. Evidence is obtained of the patency of the cystic duct, the position, size, shape, distortion of the gall bladder, adhesions and at times, of the presence of stones. It is a little too soon to estimate the amount of value both positive and negative that will be gained by this method of examination.

Attention has shifted in recent times from cholelithiasis to cholecystitis, until now some of the more extreme surgeons are removing gall bladders which show by microscopic examination only the mildest degrees of abnormality. Grossly these gall bladders are clinically recognized by some of the following signs: first, change of color in the direction of a whitening of the wall; second, fatty deposit beneath the peritoneum (Moynihan); third, swelling of the lymph nodes around the cystic duct; fourth, adhesions; and fifth, a hepatitis, especially when confined to the portion of the liver adjacent to the gall bladder. Graham (6), Heyd (7) and others have stressed the importance of gall bladders from the clinical side which are the seat of these mild changes. They believe that infections of the alimentary tract with colon bacilli or other germs are carried to the liver by the portal vein and these bacteria are excreted into the bile and then are passed through the wall of the gall bladder by the lymphatic route into the liver again thus setting up a vicious circle with a resulting hepatitis and at times with extension to pancreas, duodenum and stomach. On this view the gall bladder disease would be dependent upon some pre-existent intestinal tract infection—for instance, appendicitis or hemorrhoids. It is somewhat weakening to this point of view to find that the acute infections in the abdomen do not set up with any frequency either an acute or chronic vicious circle of this kind and that fairly severe types of gall bladder disease exist for long periods without generalized evidence of illness and only give local symptoms. It will take time to tell how clinically important these mild infections of the gall bladder may be. At St. Luke's Hospital the surgeons have cultures made of the bile and gall bladders in the majority of their cholecystectomies and I should say that in more than fifty per cent they are reported sterile. Our commonest bacterial finding is of the colon bacillus. Rosenow claims to find the streptococcus as

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a frequent invader of the gall bladder wall even when the bile is sterile and he looks upon such lesions as are found in the tonsils and roots of the teeth as the source of the infection. He thinks the streptococci show a selective affinity for certain tissues, for instance, that a certain streptococcus viridans cultivated from the human gall bladder will, if injected into an animal, set up an inflammation in its gall bladder. Other observers have not been successful in obtaining the same results with their cultures, which may be due to a less careful and elaborate technique. Zinsser (15) questions the accuracy of this work. Some of the lesions seen in the gall bladder may be partially mechanical as distinguished from bacterial in origin. These changes are in the nature of hemorrhages and oedema rather than suppurative inflammation and might be explained by large stones becoming impacted in the ampulla and interfering with venous return.

Microscopically, the mild types of so-called chronic cholecystitis are characterized by fibrosis, lymphocytic infiltration and especially by an interesting type of fatty degeneration which we see in varying degrees in the connective tissue immediately underlying the lining epithelium of the gall bladder. This change is responsible for the so-called "strawberry gall bladder." One might question whether the gall bladder of chronic cholecystitis is to be explained on the basis of bacterial infections of its wall or whether it may not be equally well explained by the action of toxins, the bacteria themselves which produce the toxins being far removed in some part of the intestinal tract.

It seems hardly likely that a sole cause will ever be found which adequately explains the formation of stones. At the present time, the conditions which, we think, have a bearing on their presence may be grouped under three headings:

First, stasis. It is undoubtedly true that gallstones occur with more frequency in women who are fat, past the most ac-

tive period of life, and are in general of the lethargic type. As has been intimated above under the physiology of the gall bladder, a sort of stasis with resulting concentration of bile is a normal condition and it is not difficult to think of this concentration as being carried at times to a too great degree. At any rate the gall bladder rather than the ducts or liver is the usual place for the formation of stones though not invariably. Gallstones are almost unknown in uncivilized races and among the very civilized early Egyptians, they were equally unknown; but there is little doubt that the average life among the Egyptians was much more active and out-of-doors than among our civilized selves. The civilized Chinese suffer frequently from gallstones. Stasis can hardly be thought of as a more than contributing cause.

Second, the metabolic theory. This seeks to explain the formation of stones on the basis of an increased cholesterin content of the blood. There are a considerable number of conditions such as typhoid fever, pregnancy and chronic visceral disease which are known to increase cholesterin in the blood. According to Aschoff (10) the gallstones of this type are always single. On section they show a radial and crystalline structure and may later be coated with other materials. He says when there are many stones in the gall bladder he never finds more than one of this pure cholesterin type. Chauffard (8) has examined the nuclei of these stones and says they show no reaction for proteid nor in any other way indicate an inflammatory origin.

Third, the infectious theory. Present day surgeons seem to lean to infection as a prime cause of stone. The old theory of Naunyn attributed them to a stasis in the bile ducts combined with a mild catarrhal infection of the ducts which had caused an increase in the cholesterin content of the blood. Bacteria may be eliminated in the bile without exciting any inflammation in the gall bladder (Aschoff). Furthermore, in-



flammation in the gall bladder wall does not necessarily and immediately lead to the formation of stones. Sometimes, the gall bladder is packed full of stones of the same size, shape and composition and it seems to have been assumed with reason that these stones have all originated at the same time. In the days of cholecystotomy the great majority of patients remained free from stone after operation and in fact Kehr and William Mayo thought that the recurrence of stone was pretty good evidence that stones had been overlooked at the time of the first operation. At times we remove an inflamed gall bladder which has previously been operated on by cholecystotomy, years after the first operation, and are surprised to find that it has not reformed stones. It seems then that at times the formation of stone is a sudden process in the course of an inflammatory disease. I should like to illustrate by what is known regarding typhoid carriers and their relation to cholelithiasis. In these carriers, the gall bladder is found to be the seat of an inflammatory process and is continually discharging typhoid bacilli into the duodenum for an unlimited number of years. Sometimes these patients have stones but the stones may have preceded the typhoid fever and be the partial cause of the patient's becoming a carrier. In the usual case, it is believed that the typhoid patient has a cholecystitis with his fever and remains a carrier—some for a short period of weeks or months and a few indefinitely. Probably clinical recovery usually precedes bacterial recovery. Chauffard (8) called attention to the infrequency with which patients suffer from gallstones in any reasonable time after an attack of typhoid fever. Carriers whose gall bladders have been removed within six months to a year from the beginning of their illness show bacteria but rarely stones. Pomeroy and Sheng (9) report operating on a patient for an attack of acute cholecystitis without stones 41 years after an attack of typhoid fever and finding the bacillus present in pure cul-



ture. It is true, however, that a large percentage of the chronic typhoid carriers that come to operation years after their typhoid are found to have stone. In some carriers that I have operated on for stone, many years after their attack of typhoid, it has seemed to me that the stones were of recent formation judging from their small size and the short duration of the gall stone symptoms.

Is it not possible that there is some factor essential to the formation of gallstones which acts suddenly and possibly but once in the patient's career and which is essential to their formation and that stasis, hypercholesterinemia and inflammation are but contributing factors?

The treatment of those suffering from well-defined gall bladder lesions is chiefly surgical. Hitherto antiseptics, cholagogues and other drugs have been conspicuously disappointing. The administration of bile salts seems well nigh ridiculous. It would be about as reasonable to recommend the administration of urea or uric acid to a patient suffering from renal insufficiency. Underlying diseases such as constipation, appendicitis or hemorrhoids may need treatment as also diseases of the mouth, tonsils and teeth. Probably most surgeons are not sufficiently careful about these matters. Some years ago Meltzer (2) announced that the introduction of magnesium sulphate through a duodenal tube was followed by the expulsion of bile from its duct into the duodenum. Lyon followed up the work of Meltzer and announced his method of diagnosis of diseases of the gall bladder and bile passages. He introduced magnesium sulphate through a duodenal tube and extracted the bile as it flowed into the duodenum, dividing it into three portions. The first was said to be light in color and came direct from the common duct. The second sample was thicker and darker and came from the gall bladder. The third sample was clear like the first and believed to represent bile as secreted into the hepatic ducts. Physicians with whom

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I have talked and who have employed the method have not found it as reliable as it seems to have been in the hands of Lyon.

Following this method of diagnosis, Lyon (14) and others have introduced a method of treatment of the biliary tract and other diseases using the duodenal tube and extracting large quantities of bile therewith over a considerable period of time. Lyon, in his last article, has summed up the diseases suitably treated by this method under fifteen headings and includes such conditions as cholangitis, obstructive jaundice, some cases of gallstones, postoperative hiccough, post operative ileus, persistent biliary fistula, gall bladder catarrhs, headaches, migraine, some asthmas, some cases of neuritis and arthritis, and even epilepsy. It is difficult to see why magnesium sulphate should be so much more efficient given through a duodenal tube than when given by mouth on an empty stomach. Furthermore, it is hard to understand that there should be great benefit from removing one's bile through a tube in preference to allowing it to pass in normal fashion. Carlson (13), in commenting on Lyon's paper, fails to follow his physiology and says he knows of no evidence tending to show that the discharge of one's own bile into one's own duodenum is deleterious. At any rate, one can hardly believe that such a method of treatment can be effective in the presence of the severer forms of gall bladder disease.

In considering the future of patients suffering from a well-defined gall bladder disease or stones, there are a number of factors that come under consideration on some of which we have rather accurate information and on others very inaccurate. With regard to the latter there is the question as to how many of the patients who have gallstones do or will have symptoms referable to the stones. In the past, we used to hear rather frequently of "silent gallstones" as though many persons carried them to the grave without ever being

inconvenienced by them. Some surgeons, on the other hand, would deny that gallstones ever remain for a considerable period without giving symptoms. On the other hand, I have seen it stated in two recent publications that probably ninety per cent (11) (12) of the gallstones give scarcely any symptoms of their presence. If the statement were made that ninety per cent of them left without operation would not cause death, one might consider the question debatable; but that the patients with any such frequency go through life without discomfort is not at all likely. I think the impression that there are innocuous gallstones has been largely obtained by consideration of autopsy reports of almshouses and homes for the aged. My own impression is that the stones nearly always give symptoms either constant or intermittent but that very often the physician is not expert enough to recognize that the symptoms are due to gall bladder disease or that the patient does not consult a physician for what he considers himself competent to diagnose as indigestion. In routine laparotomies, a large part of which are for pelvic diseases in women, it is our habit in these days to palpate the upper abdomen and we frequently encounter an unsuspected gallstone. If one goes over the history again in these cases after the operation, he will usually find that the patient has had symptoms which could be more easily explained on the basis of a gall bladder disease than of the pelvic condition for which operation was undertaken. Hence I think it is the habit of surgeons of the present day even in the absence of present symptoms to advise operation in cases of gallstones when age and infirmity do not make the operation hazardous. In the uncomplicated case, the operation may be considered very safe in patients under fifty years of age but the outlook is quite different at any age in those suffering from septic cholangitis or obstructive jaundice.

The operation of cholecystectomy has largely replaced the

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operation of cholecystotomy with drainage. With many cholecystotomy is only employed under unusual conditions or in desperate cases. We cannot deny that there is an occasional case in which the drainage operation would be the safer for the patient and it is not good judgment to promise a cholecystectomy when advising operation. In recent years I have done but one cholecystotomy to twenty-five cholecystectomies. Cholecystectomy has been the routine in acute and suppurative types of inflammation and my fatalities have not resulted from employing it in such cases. Judd has recently expressed the same opinion. At Roosevelt Hospital Cave (17) reports 105 cholecystotomies which seem to have been done in the emergency type of cases but says that half of these cases required a second operation.

In a series of 250 cholecystectomies my mortality has been 2%. Whipple, of the Presbyterian Hospital, reports a series of over 100 cholecystectomies without a death. Judd (16), reporting on the cholecystectomies at the Mayo Clinic for the year 1924, reports a mortality of 1.6%. Cave (17) collected the statistics of Roosevelt Hospital for a period of sixteen years, the operations being performed by a considerable number of surgeons, and reports 30 deaths in 470 cases, a mortality of 6.3%. A comparison of mortality in different series is hardly fair unless we take into consideration whether the series is a recent one, whether the cases are selected and whether a large number of nearly normal gall bladders are removed. The figures above quoted represent mortalities in simple cholecystectomy but do not apply to common duct surgery. We might generalize briefly by saying that no matter who the surgeon be he will probably have four times as high a mortality in common duct surgery as in that of the gall bladder alone. My mortality in 71 cases has been 12% but I hope with our means of combating hemorrhage and sepsis, it may be considerably reduced in the future.

Until very recently surgeons have reported largely their hospital mortality in operations and have not followed their cases over long periods of time and we know that it is difficult with our shifting population in large cities to follow our post-operative cases. This has been partially remedied by establishing a follow-up system in the hospitals. At St. Luke's Hospital, ours has given us valuable information for a period of ten years. The New York Life Insurance Company recently wished to investigate one surgeon's results and abandoned the investigation when they found that they were unable to trace even 50% of the patients. The investigation was abandoned because of an uncertainty as to whether the death rate of those whose records could be followed would be the same as among those who could not be traced. I am greatly indebted to the New York Life Insurance Company for the interest and energy which they have shown in following my cases. I submitted to them my own records of 350 cholecystectomies. In addition, many of these patients had had operations on the common duct but no cases were included of operations on the common duct alone such as cases of stricture of the common duct and secondary operations for removal of stone in the common duct when the gall bladder had previously been removed. Many of these patients in addition to the gall bladder surgery had hysterectomies, appendectomies and gastro-enterostomies. The information was obtained by the insurance company in a variety of ways—from my office records and those of St. Luke's Hospital, by telephone calls, from one or more letters written to the patients, from the Bureau of Vital Statistics and through inspections made by the insurance company. The tracing of women patients is made more difficult because of the changing of names through marriage.

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Among the 350\* patients, there were found to be 441 deaths—13 among men and 28 among women. Analysis showed that there were 4 women patients to one man but after considering the age distribution, it was found that the mortality among men was about the same as that among women and accordingly the data for men and women were combined. During the year of operation there were 23 deaths among the 235 cases which were traced and used for study. This is a death rate of 96 per thousand. It seems fair to assume that the deaths following operation and those occurring soon thereafter would be known. Assuming this to be so, it would be fair to assume that the 23 deaths mentioned during the first year were all the deaths among the 350 traced and untraced patients. This would give a rate of 66 per thousand. The former rate is undoubtedly too high. In determining the relative mortality, the thing which interests the insurance physician as distinguished from the operating surgeon, a measuring rod must be found. Insurance companies measure their mortality with relation to one or the other of the standard tables based on their experience. The lives, however, from which these standards have been made up have all been examined for life insurance which is not the condition of the group under review. It would seem, therefore, desirable to use another table, the most suitable of which is based on the population of the United States and known as the "United States Census 1910 Table." If the 350 individuals, under review, had had a mortality during the year equal to that among the population as a whole, there would have been five deaths whereas there were twenty-three, including those dying post-operatively. This mortality does not seem to be higher than would be expected.

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\*These patients constitute a consecutive series operated on in the 10 years beginning January, 1916.

After the first year it is rather difficult to make a very positive statement on the matter because of the considerable number of patients that could not be traced. If it were assumed that the cases not traced had the same mortality as those whose records are known, there would have been 29 deaths. The death rate among a similar group of people in the population at large would have been theoretically 82.4%, so that the mortality after the first year following operation was practically the same as among the population of the United States measured by the 1910 census. Taking these facts at their face value, it might therefore be stated that excluding the first year after operation, the mortality was about the same as of the population as a whole but the mortality has distinctly improved since 1910 for which due allowance should be made. The statement, however, involves the assumption that the mortality among those not traced was the same as those traced. If we assume that the mortality was twice as heavy among those not traced, the mortality after the first year following operation was 40% higher than among the population as shown by the 1910 census.

The only safe conclusion which we can draw is that the mortality after one year following operation is higher but not much higher than among the population as a whole. We cannot draw any practical conclusions from the causes of death as these are not accurately given in the majority of cases. I am indebted to Dr. Charles I. Harris, Commissioner of the Department of Health in New York City for permission to use the records of the Bureau of Vital Statistics. An attempt was made thus to trace 120 cases not otherwise accounted for and 5 deaths were found recorded. It was, of course, impossible to say how many of these 120 may have changed their residence or how many women may have changed their names. It might be fair to assume, perhaps, that the deaths did not occur within a year or two after the

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operation as otherwise the information would have been available.

### CONCLUSIONS

1. Recent years have added much to our knowledge of liver and gall bladder functions both in health and disease.
2. Cholelithiasis is the condition which chiefly demands gall bladder surgery and this surgery is curative in the great majority of cases.
3. Cholecystectomy for milder types of inflammation without stone has not proven very satisfactory.
4. Cholecystectomy has very largely supplanted cholecystotomy. Neither operation *per se* has any great dangers. The mortality is contributed by the disease for which operation is undertaken and associated visceral diseases.
5. Life expectancy of a patient requiring cholecystectomy is considerably reduced in the year of operation.
6. In following years, the patient who has survived cholecystectomy has a life expectancy very little less than other persons of the same age.

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At the conclusion of the reading of the paper, Dr. Matthews presented slides illustrating points brought up in the paper:

1. Several slides were shown of X-ray photographs of gall bladders removed at operation and containing stones. These were shown to illustrate how nearly the shadow of a stone may correspond in density to that of the gall bladder itself containing bile. This illustrates the difficulty of an attempt to photograph gall stones while still in the body.

2. Several radiograms of patients in whom gall stones were easily recognized either as solid or ring-like shadows. In each of these patients it was said that the diagnosis had been evident by the history without resort to X-ray.

3. Slides to illustrate the association of duodenal ulcers and gall stones, an association which has often been called to the attention of the profession. It was said to be too frequent to be accidental. The speaker had found them associated in ten per cent of his duodenal ulcers.

4. Numerous slides were shown to illustrate the methods of cholecystography as developed by Graham. The speaker called attention to cases in which the method had been of positive and others of negative value in diagnosis.

5. Slides were shown of abdominal viscera indicating the various theories in which the gall bladder or liver constitute

a part of a vicious circle of infection as elaborated by Lyon, Graham and others.

6. Photographs of a good illustration of the strawberry gall bladder, a condition characterized by a lipoid degeneration of the connective tissue immediately underlying the epithelium of the gall bladder.

7. Slides presenting tables of results of gall bladder surgery. (A) 250 cases of cholecystectomy with a mortality of 2%. (B) a list of the causes of death in the fatal cases. (C) Table showing the four times greater mortality in patients requiring common duct surgery. (D) Table of cases of gall bladder surgery with pure culture of typhoid in the gall bladder.

8. Slides illustrating the specimens removed and diagram describing the operation in the case of a patient who had the gall bladder removed for stones 20 years ago and who was well for the 19 succeeding years. During recent months has developed symptoms of common duct infection and at operation was found to have common duct and intrahepatic stones.

Dr. Mathews said in closing that the mortality from gall bladder operations is very small in patients under 50 years of age, and Dr. Rowley has called attention to the mortality resulting from visceral infirmities which complicate our work and which are chiefly a menace in patients over fifty years of age. The chief danger is contributed by associated lesions and infections. He said it is not disputed that mild types of gall bladder change are associated with hepatitis but it was his opinion that there is room for debate as to whether the hepatitis results from the gall bladder condition or the gall bladder condition is secondary to the hepatitis or whether the lesions in both are not toxic and the result of absorption of bacteria or toxins from the intestine. It was his

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opinion based on his clinical experience that no great benefit results from the removal of the slightly diseased gall bladder. It may be urged by some that this only means that his selection of cases has been bad. He called attention to the infrequency with which acute appendicitis, acute or chronic gall bladders which are conspicuously thickened and infected and draining pus into the common duct over long periods of time, give rise to any severe type of hepatitis. He had been greatly interested in the follow-up of the life insurance company on his own cases and thought it would be greatly to the advantage of surgeons and patients if they thought of their work more in terms of life expectancy rather than in terms of immediate mortality. He commented on the cases of typhoid gall bladder, on the length of time infection continues, that the bacteria are constantly present in the gall bladder as well as their products, that the typhoid gall bladder is not uniformly associated with stone and that these persons seem to be clinically well and in particular are not suffering as the result of any marked secondary lesion in the liver.

Dr. Hobbs—Gentlemen, we have heard this very interesting paper and two have consented to discuss it. After that, I am quite sure that Dr. Mathews will be very glad to answer any questions you may want to ask. I will now call upon Dr. Rowley.

Dr. Rowley—Mr. President and Gentlemen: In attempting to measure the insurance value of lives presenting a history of diseases of the gall bladder we are confronted with many factors that may make the problem not an easy one.

In 1908, Dr. Symonds presented to this association a very carefully prepared report based on a scientific study of the experience of the Mutual Life Insurance Company in cases with a history of hepatic colic or gall stone—being careful

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to exclude the cases of doubtful diagnosis—and the mortality experiences shown was 131% of the expected.

The more favorable experiences—107% of the expected—in the Specialized Mortality Investigation was believed to be due to the inclusion of a number of cases without true hepatic colic or gall stone history.

The very excellent paper by Dr. Homer Gage, in 1910, gave the members of this association the benefit of his own views on this subject and it embraced the judgment of other masters of surgery at that time. Dr. Gage's paper, based on his wide experience as a surgeon and a medical director, pointed the way to a more intelligent selection in these cases.

Dr. Charles H. Mayo, in 1916, addressed us on the subject of "Life Expectancy Following Operations on the Gall Bladder" and one of the observations mentioned by him was that while the conditions in the gall bladder are not often in themselves fatal, the patients die of secondary degenerations.

Dr. Mathews has given us in his splendid paper not only a resume of recent research on the physiology and pathology of the gall bladder but a study of the later results in his series of operated cases, presented in a way that gives information of especial interest to us.

In the study of this group of cases presented by Dr. Mathews, one is led to ask:

- (1) What was the period of observation of the cases traced after the first year?
- (2) What was the age distribution of those cases?
- (3) Was overweight a predominant characteristic in this group of cases?
- (4) Were the cases requiring hysterectomy, appendectomy, or gastro enterostomy sufficiently numerous to materially influence the results?

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Our actuaries have concluded from the figures given by Dr. Mathews that the period of observation was probably about five years, on the average.

The age distribution is important on account of the lessened ability of repair of cell damage in the older age groups, the operative mortality being very much higher above age 50 than it is below that age.

Gall stones and gall bladder affections, according to common observation, are inclined to be accompaniments of sedentary habits and overweight.

The conditions, necessitating gastro-enterostomies and hysterectomies, rather suggest something more serious from an insurance standpoint than do affections limited to the bile tract.

We may assume from Dr. Mathew's figures that the period of exposure after the first year was on the average from age 51 to 56. For this age group the mortality in the general population tables of 1910, which he found to be the same as that of the cases that he traced, is about 120% of the mortality in the American Men Ultimate table. To subject these cases to the process of insurance selection would probably produce a group having the same mortality as the general population after the same process of selection and it would appear, therefore, that these cases are reasonably normal risks.

While it seems justifiable to assume that the influence in producing an extra mortality is a decreasing one with the lapse of years, we are unable to say when that influence ceases. For practical purposes, however, we may conclude that the class is a reasonably good one from an insurance standpoint after the first year has passed, with freedom from all signs of possible relation to the original trouble.

And here we are confronted with some uncertainties inasmuch as an insurance report not always gives a full ac-

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count of the symptomatology that may have intervened. Like the small boy who had eaten too many green apples and was told by the Christian Scientist that he had no pain, said, "I have inside information," so with these applicants. While the history as we see it recorded may be clear, they sometimes have "inside information" that would be of interest to us.

The return of symptoms may be due to further trouble within the biliary system or to infection elsewhere within the portal vein area of drainage.

There is a certain measure of comfort to us in Dr. Matthews' view that the secondary or associated intra abdominal infections do not, with any frequency, set up either acute or chronic disturbances of an important nature. Their true significance, however, may yet become better known through more extensive bacteriological and clinical observations.

It is generally accepted now that an infection in the gall bladder is practically always accompanied by a hepatitis of greater or less degree, and it would seem most likely that the infection takes place through the lymphatic route.

In turning back a few years, we find that Adrian (1) in 1901 was among the first to conclude that appendicitis was a blood infection.

We also find that Ochsner (2) in 1906 was among the first to observe the relation between appendicitis and gall bladder disease in his report of four cases of pyloric ulcer, two of which were associated with biliary tract infection, two with appendicitis (one case having both lesions) and 13 cases of biliary tract infection, of which four had pyloric ulcer and seven appendicitis.

MacCarthy and McGrath (3) in 1910 reported a very thorough pathological study at operation and at autopsy. In 52 ulcers, 26.9% were associated with chronic appendicitis;

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175 cases of biliary tract infection showed almost 50% associated with chronic infection of the appendix.

To view the matter from another angle—of 2,000 chronic appendices 8.7% had biliary tract infection. From about that period the coincidence of these lesions was given increasing attention and numerous reports of surgeons, as well as hospital statistics, lent testimony to this close relationship.

LaRoque (4) in 1913 in stating the source of bacteria in the causation of pyloric ulcer, added: "Pyloritis, pyloric ulcer, bile tract infections, pancreatitis, and perhaps cirrhosis of the liver, are late results of infection, primarily located most commonly in the appendix, though in many cases in some other regions drained by the portal vein; and these lesions frequently exist together in the same patient at the same time or may follow each other in rapid or slow succession."

What appears like the view of an extremist is that attributed to Moynihan (5) in his belief that the appendix should be removed in 90% of cases of ulcer and biliary disease.

1910 and 1911 of 322 ulcers operated upon he discovered 111 chronic appendices, requiring appendectomy.

Deaver (6) in 1919 stated that in case of gall bladder infection, whether there are calculi or not, operation should include removal of the appendix, and examination of other abdominal viscera for associated disease.

I am not certain but these views may be a little too extreme for general acceptance by other leaders in surgery but, nevertheless, they serve to point out to us in insurance medicine the possible pitfalls that may attend the afterhistory of cases with gall bladder infection.

Whatever may be the true value of the gall bladder in human economy, there is abundant evidence that individuals



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can get along without it and suffer no great sense of loss other than financial.

Nature, in following her custom in display of lavishness, has provided us with an abundance of small structures along the bile ducts which histologically resemble the gall bladder and are referred to by Beall (7) as supplementary little gall bladders, and it would appear from the experimental work of Sweet (8) that these sacculi or supplementary gall bladders undergo hypertrophy after cholecystectomy. Through the rich lymphatic connections these supplementary gall bladders participate in all of the infections of their big brother, but who wants to contemplate several hundred, perhaps thousands, of infected gall bladders in one person at the same time!

Dr. Mathews has called attention to the large amount of experimental work done in the past few years with the object of learning the function of the gall bladder and while much is still to be learned, it seems justifiable to dismiss the idea that the gall bladder is to serve as a storehouse for bile in the intervals between the demands for active digestion.

In seeking some suggestion of the function of the gall bladder in a comparison of some species of lower animals one encounters a number of inconsistencies, but according to Sweet (8) "The true meat eaters possess gall bladders, while the varieties lacking this organ are, in the main, herbivorous. The main element present in a meat diet and not found in a vegetable diet is animal fat; and certainly the fat element of the diet is almost entirely lacking in grass eaters." As Dr. Mayo said, "There are a few humans with congenital absence of the gall bladder and they resemble the ass."

In line with the relationship between the gall bladder and fat metabolism the experimental work of Sweet (8) has shown pretty clearly that the removal of the gall bladder exerts a definite effect upon the metabolism of the lipid, cholesterol

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—an immediate rise in blood cholesterol to almost double the normal following cholecystectomy.

The less frequent return of symptoms necessitating a second operation has led surgeons in the last few years to remove the gall bladder rather than drain it, except under exceptional circumstances.

What about the insurability of cases where drainage only has been done? I realize this is a bit outside the scope of Dr. Mathews' paper but it may be of interest briefly to record that in Guys' Hospital Report of April, 1925, a study of 425 cases treated between 1895 and 1915 showed that 9% of cholecystotomy cases required another operation.

Dr. John W. Deaver (9) in 1920 reported the necessity for re-operation in 10% of cholecystotomies—and in 1.3% of cholecystectomies.

Reports from the Mayo Clinic apparently are quite similar.

Branon (10) in 1920 studied 425 cases of gall bladder disease treated at the Hartford Hospital from 1914 to 1918 and found that 8.2% had previous cholecystotomy.

In 888 cases of biliary tract disease treated at the Johns Hopkins Hospital from 1889 to 1924, Blalock (11) reported 80 cases in which more than one operation had been done, and in these 80 cases 11% had had the gall bladder removed and 89% had had the gall bladder drained.

As to the time elapsing between operations—in 56% of the cases it became necessary in less than one year; in 86% of the cases it became necessary in less than five years.

My own guess is that cholecystotomy cases will show a mortality experience less favorable than that of cholecystectomy cases and that they should be subjected to a period of probation longer than one year for insurance purposes.

Dr. Mathews, in his splendid paper, has given us so much that should be of practical value to us in our work that

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after fully digesting and assimilating it, I am sure that we shall be able to deal more intelligently and fairly with gall bladder cases that apply to our companies for insurance, and I personally feel that the association is greatly indebted to him for this valuable contribution.

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11. Blalock, Alfred, Johns Hopkins Hospital Bulletin 406; Dec. 1924.

Dr. Hobbs—Dr. McCulloch.

Dr. McCulloch—Mr. President and Gentlemen: There is absolutely nothing that I can add on this subject presented here so ably by the essayist. His investigation was conducted under the most ideal circumstances. His "holy alliance" with the follow-up system of the insurance company which helped him in this matter is unusual. I think that Dr.

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Mathews is deserving of further consideration from the insurance company. They should place at his disposal also their collecting department in order that the unpaid accounts in these 250 cases on his books may be gathered into the fold by their efficient collection operating machinery.

Bishop Butler said in discussing this quadrant of the abdomen that it is the *terra incognita* of the human body. So many things can occur in that particular region so closely allied in their symptomatology, so far reaching in their correlation, that any man before he inserts a scalpel below the lower right costal margin may well wonder what he is going to find. But thanks to developments in diagnostic procedure, particularly X-ray, we have a much clearer understanding; and perhaps in the ten years or so that are ahead of us, there will be some very great developments, not only in what is found pre-operatively, but in anticipating what may occur eventually, and thus, by early operation benefit the insurance companies by anticipating the late results in adjoining organs.

One cannot but be impressed with the rapid change that is going on in our profession, not only as it affects the profession but as it affects the insurance business. When I got on the train to come here, I picked up the paper and found that Dr. Coolidge has discovered a ray of absolutely unknown but enormously important significance, and who can tell but what after it has been harnessed properly and studied properly and directed properly that many of the infections which we find in various parts of the body, and in this particular place as well, may be subject to a reparative service. We all feel that there is something behind it of real future value, just as it was when the X-ray first was known.

And then listening to the President's paper yesterday, I could not help recalling that the day before I left my home town, over 150 cases of paresis were reported by the Super-

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intendent of the Indiana State Hospital for the Insane which had been symptom-free and apparently recovered, previously hopeless cases. For 18 months, 26 per cent of those cases had been symptom free and apparently well by virtue of inoculation with malaria germs at the last moment.

What a threshold we are standing on! And it is papers such as Dr. Mathews has put before us that make us very hopeful. As far as our mortality is concerned, the tendency, I think, on this particular type of case is for the mortality to improve. We all know the tendency of the surgeons now to reach for the gall bladder. Perhaps there is a little backwash at the present time, but the surgeons whom I know are enthusiastic about cholecystectomy. If they take these cases earlier and earlier, we may look, I think, for a better showing and figures in gall bladder cases. They *will* be taken earlier, instead of waiting until the gall bladder is so badly affected that the adjoining organs are also involved.

In closing, I can only say that when the point is reached that a man with a typhoid gall bladder, who feels perfectly well himself is willing to get up on the table and have it removed for the sake of his neighbors, "Greater love hath no man than this, that he will give up his gall bladder for his fellow men."

Dr. Fisher—Mr. President, I have some data here that may be of interest in connection with this subject. It will only take a few minutes to present it.

We have insured 16,791 cases, issues from 1885 to 1908 carried to 1915, and 14,446 cases, issues from 1906 to 1915 carried to 1920. In both periods the mortality was several points below the general average of the company. That is where they have had typhoid fever within ten years. We insured 2,636 cases, issues from 1885 to 1908, carried to 1915; and 9,285 cases from 1906 to 1915 carried to 1920, with

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✓ a mortality slightly below the general average of the company. Cases of appendicitis without operation, last attack was in ten years. Number of cases, 1,295, issues of 1885 to 1908 carried to 1909, and 2,103, cases issues 1906 to 1915, carried to 1920. The mortality in both groups was below the general average of the company.

Dr. Hobbs—The contribution of Dr. Fisher rounds off the papers we have already had. The paper and comments of Dr. Mathews and the remarks of those who have discussed it will give us a better idea of the subject and give us a basis for better selection of these risks. I wish to thank Dr. Mathews and also those who have discussed the paper.

We will now pass on to the next item on the program. We will now hear from Dr. Rogers and Mr. Hunter on the subject of "Systolic and Diastolic Blood Pressures and Pulse Pressures Higher than the Average for Age."

Mr. Hunter: May I say a word with regard to Dr. Mathew's statement concerning the interpretation of statistics prepared by certain physicians who had evidently little knowledge of mathematics. I was recently interested in studying the mortality from cancer of the breast and found in reading different sets of statistics apparently on the same basis, that one physician reported approximately 50% of the women were alive at the end of 5 years, while another report showed only 15%. On investigation it appeared that the former had taken all the women on whom he had operated in the preceding 5 years and therefore they had been under observation only 2 1/2 years on the average, while the latter had not considered any person unless they had been under observation for 5 years after the operation.

In connection with the statistics of surgeons, I found it difficult to get permission from them to analyze and publish their data. I have wondered whether an obstacle does not exist in the fear that the results of their work may not be

apparently as good as some other surgeon. They may be afraid that their reputations may be affected. It is entirely conceivable that one physician may have very serious cases referred to him while another may not have such a high percentage of such cases, and while the former may be more capable his death rate would be higher than that of the latter.

One of the difficulties that actuaries have to face at the present time is to find some basis for comparing the mortality among different types of insured lives. Medical Directors realize that prior to 20 years ago the mortality was higher than in the succeeding 15 years, and that in the past 5 years we have had the lowest mortality that has ever been experienced in this country. In fact, 20 years ago, if an actuary had prophesied the present low mortality, he would have been considered too optimistic for safety. The result of the varying mortality, including that due to the influenza epidemic of 1918 and 1919, is that it is difficult to determine the relationship between the mortality among substandard and standard lives. The period for which the investigation is made must be taken into account in determining the standard mortality to be used in comparison with substandard risks. To convey to the readers a clear conception of the mortality among special classes it is no longer satisfactory to make a comparison with the expected deaths by the M. A., or the American Men Table. The only way in which the reader may obtain such a conception is to be told the mortality in the class under investigation compared with the mortality for the same period in the company on standard risks.

One of the chief features of the paper which Dr. Rogers and I have submitted is the higher relative mortality at the middle than at the younger and older ages at entry. This puzzled Dr. Rogers and myself a great deal and we made a number of tests to determine the accuracy of our work.

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Until a larger amount of material is available we can only make conjectures regarding the cause for this condition. There is one which has not been mentioned by us and which is of an actuarial nature. We have compared our experience with the American Men Select Table but our improvement in mortality in recent years on "standard" risks has been greater at the younger than at the middle ages. It follows, therefore, that if the experience on the blood pressure cases, treated as substandard, were compared with the experience of our company on standard risks in recent years the ratios would be higher at the younger and lower at the middle ages at entry than those given in our paper. After making such an adjustment the mortality at the middle ages is still much higher relatively than at the younger or older ages.

In the early days of taking blood pressure readings the systolic was considered the main factor in mortality. Then, soon after we commenced to take the diastolic, it was contended that the diastolic was of much more importance than the systolic. Later a few persons seemed to think that the pulse pressure was of considerable importance. Dr. Rogers and I, as a result of our recent investigation, now believe that the systolic is the dominant factor, and that the pulse pressure is not in itself a matter of significance, but is merely the difference between the systolic and the diastolic readings both of which are of significance. Our present opinion of the pulse pressure is largely the result of the present mortality investigations, only one-third of which have been published. None of the investigations of pulse pressure alone showed satisfactory results.

May I warn you that our results are based on substandard risks and should not be compared with a specially selected group of risks with the same improvement on which standard insurance was issued. It is generally possible to pick out a small percentage of the best substandard risks with slight



or moderate impairments and obtain a satisfactory mortality, but such statistics are not a safe guide for other companies with a different method of selection, or who take such risks more freely.

We have frankly laid down our findings so that you are in a position to come to your own conclusions. We believe that high systolic or high diastolic blood pressure is more serious than we had anticipated and it is likely, therefore, that our ratings will be raised in the near future although that may mean the loss of some of our business to other companies. If we feel that the ratings should be increased for any impairment it is our practice to do so, irrespective of what the results will be in the matter of new business.

I thank Dr. Rogers for his courtesy in requesting that I speak ahead of him and I am sure that he will have more of interest to say than I can, as a layman.

Dr. Rogers—Mr. President and Gentlemen: I think that most of you must have been impressed with what my colleague, Mr. Hunter, has said and have noticed how much he has profited by the training in medicine to which he has been subjected during recent years. Mr. Hunter has told you that we were a great deal puzzled to explain one of the results of our study of high blood pressure, that is to say, the high mortality in the decade 40 to 49 inclusive. Indeed, it is fair to say that we were so much perturbed about it that for a time we rather doubted the correctness of our own figures. Some of you will remember that several years ago we made a study of the mortality in systolic hypertension. In this present study, we used a certain amount of the material which we had used in our former study but, on the whole, they covered such different periods of time that we were very anxious to make a comparison of our old findings with these that we now submit, to see if our earlier statistics revealed the same state of affairs. Unfortunately, the work-

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ing sheets of our former study had been mislaid and at the time the galley proof of our papers was prepared we had no means of determining whether this bulge in mortality was peculiar to our later study or occurred in the earlier as well. However, we have since then discovered the working sheets of our earlier study and I cannot tell you how well pleased we were to find that the same state of affairs was disclosed in them. We feel bound to believe, therefore, that there must be something in the conditions themselves and not, as we feared, in our statistics, to cause the strikingly high mortality in the decade fortieth to fiftieth year of age. We are thus led to speculate whether it might not be true that simple hypertension, that is to say Albutt's hyperpiesia before it reaches the cardio-vascular-renal stage, about which we hear so much nowadays, that pure hypertension is a phenomenon which develops in the great majority of cases between the fortieth and fiftieth year. It is apparently quite sharply localized in that period of life. It would be most interesting if other companies would take up this problem to see if they also have experienced the high mortalities we have twice found in our investigations.

This leads me to add a few words about the causes and treatment of high blood pressure, which would seem hardly germane to the discussion of this subject from a life insurance point of view. Several years ago an extended study was made at the Battle Creek Sanitarium of different food stuffs, with a view to ascertaining their effect upon the reaction of the urine. Blatherwick did this work at Battle Creek, and later continued it at Santa Barbara under Dr. Sansum. It was Dr. Sansum, I believe, who first suggested that high blood pressure might be due to the excessive use of acid-ash foods and he carried on an extensive series of experiments with rabbits to test out his theory. He found that, by feeding rabbits with acid-ash food, he was able to

induce in them high blood pressure, renal irritation and marked changes in the blood vessels and that, by changing the rabbits over to a basic-ash diet, these symptoms were relieved. It may be proper to define here what is meant by acid-ash and basic-ash foods. All foods which are oxidized or burned up in the tissues of the body leave behind them ashes of one sort or another, just as wood, when burned on the hearth, leaves behind it basic-ashes. At Battle Creek and at Santa Barbara the nature of the residual ash of many articles of food has been determined and constant application is being made at those institutions of the results of their observations. Every article of food then, which is taken into the body, leaves behind it its ash, whether acid or neutral or basic, depending upon the nature of the food, and high blood pressure cases are relieved in a most striking manner when the patients are confined to a basic and neutral ash diet. I am led to make this statement for the reason that, during the last eighteen months, I myself have applied Dr. San-sum's theory in a number of cases of high blood pressure and always with quite remarkable results. Not only is the principle which I have here tried to enunciate applicable to high blood pressure, but it is strikingly useful in cases of chronic Bright's disease, in which a bland, low nitrogen diet is important. Indeed, I have come to feel confident that it is a great deal more than an even chance that any case of high blood pressure will be greatly benefited by the exclusive use of basic and neutral-ash foods. I have been giving quite widespread publicity in the *New York Life* to this plan of treatment of high blood pressure and I shall be very glad, indeed, to give any of you who are interested the details of it.

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### MORTALITY STUDY OF IMPAIRED LIVES NO. 5

BY DR. OSCAR H. ROGERS AND MR. ARTHUR HUNTER

#### *Systolic, Diastolic and Pulse Pressures Higher than the Average for the Age*

This is a study of the mortality among risks insured as substandard by reason of high blood pressure. An advance in age has been charged in all cases. We have made use only of the policies issued from 1916 to 1924 inclusive, carried to their anniversaries in 1925 for the reason that the practice of taking the diastolic had not become general among our medical examiners until the year 1916 and we wished to make a study of the diastolic and pulse pressures as well as of the systolic. Much of the material for the study of systolic pressures appears also in the study of diastolic, in fact, it may appear in all three investigations.

In every case the blood pressure was observed on two or more occasions and was found to be consistently above the average. If it were higher than the average at first but approximately average on several later observations, the applicant was treated as a standard risk. It has been our practice, on request within a reasonable period of time, to review high blood pressure cases and where the abnormality has cleared up, to reduce the premium to that for the true age. This practice has had the effect of rendering each of our groups slightly more substandard through the process of culling out some of the best risks. While most of the cases in this investigation were looked upon as impaired risks solely on account of abnormally high blood pressure there were included a number with minor impairments in order to increase the material, but the effect of including them cannot have influenced the result by as much as five points.

The material is of such recent origin that the average period of exposure was but little more than three years.

Partly on that account and partly on account of the comparatively small amount of it the mortality was studied by policies only. Unfortunately there is no published table which represents the mortality in our Company during the years covered by the investigation, 1916 to 1925 inclusive. While we have employed the standard generally used at present for measuring mortality—the American Men Select Table—we wish to point out that our Company's general mortality is distinctly below that table. Accordingly the ratios of actual to expected deaths hereafter given are lower than would have been the case if we had been able to use our own experience as a standard. Moreover, the improvement in our Company's mortality has been greater at the younger than at the older ages. In interpreting our results, due allowance should be made for these facts. As was to be expected the average age of the cases treated as substandard on account of blood pressure was distinctly higher than that for all entrants—indeed it was nearly ten years higher. The expected deaths had been calculated at the true age at issue and not the rated age.

The average blood pressures, whether systolic, diastolic or pulse are practically those used by the Joint Committee on Mortality in their recently published report on Blood Pressure.

Under ordinary conditions of health and environment these pressures, systolic, diastolic and pulse, stand in the relation 3, 2 and 1, or to be exact, in that of 120-79-41 at age 20 to 134-87-47 at age 60. These relations are fairly constant and any marked departure from them is looked upon with suspicion. It is the object of studies like this to determine the effect upon mortality of these "marked departures."

It must be remembered that of the three blood pressures, the systolic and the diastolic are determined by direct observation, while the third, the pulse pressure, is only the difference between the other two. Theoretically it should be pos-

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sible to have nine combinations of the two blood pressures, systolic and diastolic, as follows:

Systolic high with diastolic high

“ “ “ diastolic average

“ “ “ diastolic low

Systolic average with diastolic high

“ “ “ diastolic average

“ “ “ diastolic low

Systolic low with diastolic high

“ “ “ diastolic average

“ “ “ diastolic low

In practice, however, the most general of these is the normal group in which the systolic and the diastolic are both average. In the abnormal groups, the most common in practice is that in which both the systolic and the diastolic are high. Next in order of frequency is a high systolic with an average diastolic. The other groups are infrequently met in practice. It is evident, for example, that a low systolic with a high diastolic would result in a pulse pressure so low as to be outside the limit of health.

Another matter which we wish to point out is that if the systolic is higher than the average and the diastolic is normal, the difference must be taken up in the pulse pressure. Furthermore, if the systolic is higher than the average and the pulse pressure is average, the diastolic must be the same number of millimeters above the average as the systolic, but the diastolic is usually about two-thirds of the systolic.

### HIGH SYSTOLIC PRESSURE

There were 4,214 cases in which the systolic blood pressure was 15 or more millimeters above the average for the age. These we divided into three groups according to the number of millimeters of departure from the average as follows:

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A. 2,005 cases, 15 to 25 MM above the average for the age.

B. 1,814 cases, 26 to 35 MM above the average for the age.

C. 395 cases, 36 to 45 MM above the average for the age.

A study of these three groups gave the following:

Group		MM Above Average for Age	Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths
A		+15 to +25 MM	70	53.2	132%
"	B	+26 to +35 MM	82	47.9	171
"	C	+36 to +45 MM	18	8.6	210

Three years ago we presented our experience of high systolic blood pressure for the years 1907-1922 inclusive. It may be interesting to compare it with that of the present investigation and also with that by Dr. J. W. Fisher in 1922 on risks rejected for insurance. Unfortunately the three investigations are not on the same basis. Dr. Fisher's material and ours of three years ago were tested by the Medico-Actuarial Table, whereas that of our present investigation was measured by the American Men Select Table. Furthermore the groupings by millimeters are not exactly the same. These facts should be kept in mind in drawing deductions from the following tables:

## NEW YORK LIFE EXPERIENCE 1907-1922

MM Over Average for Age	Ratio of Actual to Expected Deaths by Medico-Actuarial Select Table
+10 to +25 MM	146%
+26 to +35 MM	138
+36 to +50 MM	248

## DR. J. W. FISHER—RISKS REJECTED BY THE NORTHWESTERN MUTUAL

MM over Average for Age	Ratio of Actual to Expected Deaths by Medico-Actuarial Select Table
+10 to +24 MM	169%
+25 to +34 MM	200
+35 to +49 MM	245

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## NEW YORK LIFE EXPERIENCE 1916-1925

MM over Average for Age	Ratio of Actual to Expected Deaths by American Men Select Table
+15 to +25	132%
+26 to +35	171
+36 to +45	210

Making due allowance for the difference in the two standards and for the years of experience, the mortality in the present investigation is approximately 7% lower than in our preceding study. The results show conclusively that, for each increase of systolic pressure over the average, there is an increase in mortality.

With a view to ascertaining the influence of age in the case of high systolic pressures, we distributed our material into three age groups, 15 to 39, 40 to 49 and 50 up. The following table gives the result.

### HIGH SYSTOLIC PRESSURE

Ages at Entry	Cases	15 to 45 MM Above the Average		
		Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths
15-39	1,619	18	18.5	97%
40-49	1,408	76	33.5	227
50-65	1,187	76	57.7	132
All ages	4,214	170	109.7	155%

In Table 41, page 47 of their study of Blood Pressure, the Joint Committee gives the results obtained by them in a "High" group and also in one "above the average." The former may fairly be compared with our material, keeping in mind that we contributed 57% of the data in the "High" group. The "above the average" group does not show the same incidence by age as the "High" group but the former represents only 10 MM above the average systolic for age.

In the experience for the years 1907-1922 presented in 1923, we did not give the results by age groups. We are led to do



so now for purposes of comparison with the present experience. The three experiences are as follows:

Ages at Entry	HIGH SYSTOLIC PRESSURE		
	Nylic's 1926 Experience	Nylic's 1923 Experience	Joint Committee "High" Group
15-39	97%	113%	130%
40-49	227	230	189
50-65	132	170	112

To say truly we have been much puzzled by these results and have examined our material, both old and new, very critically to make sure that errors have not crept into the preparation or handling of it. We have been unable to find any errors although the results may have been affected, especially at the younger ages, by the shortness of the exposure and the relative paucity of numbers. We have been led, then, to conjecture what may be the possible explanation of the differences in mortality in the three age groups. It may be that, among the young, a high systolic blood pressure is due in the great majority of cases to emotional causes, causes of a temporary and unimportant nature; that among elderly persons it stands for a low grade degenerative process—a wearing out of the machinery of the body, and that the high mortality found between ages 40 and 50 is due to a true hyperpiesia, a disease whose essential feature is an elevated blood pressure without any other symptoms. We are offering these suggestions for what they are worth.

Whatever the cause may be, it is safe to say that a high systolic blood pressure is a serious impairment, probably much more serious during the middle period of life than at either of the extremes. We should bend every effort to learn as quickly as possible whether the heavy mortality among persons with a high systolic pressure is so markedly confined to the middle period of life as our statistics show, or whether it actually extends to the younger and older lives as well.

Of the 170 deaths in the investigation 54 (32%) were

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from heart disease and 20 (12%) from cerebral hemorrhage apoplexy, a much higher death rate than the normal from these causes.

### HIGH DIASTOLIC BLOOD PRESSURE

As the diastolic pressure stands in the relation to the systolic of 2 to 3, it was thought advisable to place the limits of the groups of diastolic pressures at about two-thirds of those of the systolic. There were 2,634 cases in which the diastolic blood pressure was 10 or more millimeters above the average. These were divided into three groups according to the number of millimeters of departure, as follows:

Group A,	1,456 cases	+10 to +16 MM	above the average for the age
Group B,	824 cases	+17 to +23 MM	above the average for the age
Group C,	354 cases	+24 to +30 MM	above the average for the age
	<hr/> 2,634		

A study of these three groups yielded the following results:

### HIGH DIASTOLIC PRESSURE

	MM Above Average for Age	Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths
Group A	+10 to +16 MM	52	38.6	135%
" B	+17 to +23 MM	41	21.8	188
" C	+24 to +30 MM	24	11.2	214

Rearranging this material according to age at entry showed the following:

Ages at Entry	Cases	Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths
15-39	919	13	11.3	115%
40-49	979	53	24.0	221
50-65	736	51	36.3	140
All ages	<hr/> 2,634	<hr/> 117	<hr/> 71.6	<hr/> 163%

It will be seen from these tables that a high diastolic pressure has, on the whole, about the same significance as a high systolic pressure, taking account of the normal difference in

millimeters between the two. A comparison of the ratios of actual to expected deaths for systolic and for diastolic pressures in the three groups already studied make this clear, thus:

# RATIO OF ACTUAL TO EXPECTED DEATHS

Group	A	Systolic	Diastolic
"	B	132%	135%
"	C	171	188
"	C	210	214

This striking agreement between the results of our studies of systolic and of diastolic pressures should cause us little surprise because, when the systolic pressure is high, the diastolic is usually also above the average for the age and, therefore, the material which entered into our study of systolic pressures formed a large part of that used in our study of diastolic pressures as well. In view of this circumstance, we endeavored to obtain groups of high systolic with but average diastolic pressures, and also groups of high diastolic with but average systolic pressures. With reference to the latter, the diastolic, we were unable to find a group sufficiently large to justify investigation, but in the systolic, we found two groups which serve to throw some light upon the subject. In group "A" of the systolic (+15 to +25 MM) we found a sufficient number with diastolic pressures between -7 to +7 MM, and in group "B" of the systolic (+26 to +35 MM) we found a sufficient number with a diastolic of -7 to +7 MM. These two groups yielded the following:

Number of Cases	MM Above Average for Age	Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths
591	+15 to +25 -7 to +7	24	16.8	143%
425	+26 to +35 -7 to +7	18	10.1	178

The ratio of actual to expected deaths in group "A" of the high systolic without regard to the diastolic was 132%. In the corresponding group in the above table it was 143%

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where the diastolic was known to be about normal. In group "B" of the high systolic the ratio was 171% without regard to the diastolic and was 178% with an approximate average diastolic. These results suggest that the systolic pressure is a dominant guiding factor.

### HIGH PULSE PRESSURE

A high systolic pressure is a well-known, a frequently observed phenomenon, and one, the underlying causes of which are coming to be understood. A high diastolic pressure also has come to have a rather definite significance, but with regard to high pulse pressure our knowledge is very indefinite and it is with respect to it that we were hoping to gain some light. After many studies, the following fairly represents the unsatisfactory nature of our results:

### HIGH PULSE PRESSURE

		Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths
Group A	+8 to +12 MM	30	20.5	146%
" B	+13 to +17 MM	45	25.8	174
" C	+18 and more	55	39.5	139
		<u>100</u>	<u>85.8</u>	<u>152%</u>

Evidently these are an expression of a mixture of dissimilar underlying causes. Analysing the cases in each of these three groups with reference to their average systolic and diastolic pressures we find:

	Systolic		Diastolic	
	Existing And Lapsed	Dead	Existing and Lapsed	Dead
Group A	151 MM	156 MM	99 MM	102 MM
" B	153 MM	158 MM	95 MM	99 MM
" C	156 MM	157 MM	88 MM	88 MM
All	<u>154 MM</u>	<u>157 MM</u>	<u>93 MM</u>	<u>95 MM</u>

Notice that:

1. The average systolic pressure in each group is higher

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among the "Dead" than the "Living." This is true also in two of the three groups of the diastolic pressures.

2. The systolic pressure in each group is much higher than the average, fully 25 MM, while the diastolic is only about 10 MM higher, showing that in our material we have to do with a large proportion of high systolic pressures.

It is quite evident that within the limits of our material the pulse pressure may not be considered alone but only in connection with the systolic or the diastolic. This point is brought out still more strongly by a study of two typical groups:

MM ABOVE THE AVERAGE FOR AGE					
		Systolic	Diastolic	Pulse	
Group A		+15 to +25 MM	0 to +9 MM	+15 to +25 MM	
		+26 to +35 MM	+10 to +20 MM	+10 to +20 MM	
Group B		Actual Deaths	Expected Deaths by A. M. Select Table	Ratio of Actual to Expected Deaths	
		23	16.2	142%	
Group A		36	20.4	176%	

The group with a pulse pressure of from 15 to 30 MM above the average indicates a distinctly lower mortality than one in which the pulse pressure was only 10 to 20 MM above the average. In our study of high systolic pressure the mortality in the group +26 to +35 MM was 171% and in our study of diastolic pressure the group +10 to +16 showed a mortality of 135%, whereas in Group "B" of this study, which combines both of these conditions, the mortality is 177%. Evidently the high systolic pressure was in this case the cause of the high pulse pressure.

By putting side by side the figures from the two preceding tables an interesting sidelight may be thrown on the importance of the diastolic pressure, as follows:

Systolic	Diastolic	Ratio of Actual to Expected Deaths
+15 to +25 MM	- 7 to + 7 MM	143%
+15 to +25 MM	0 to + 9 MM	142%
+26 to +35 MM	- 7 to + 7 MM	178%
+26 to +35 MM	+10 to +20 MM	176%

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While the foregoing indicates that the mortality is practically independent of the diastolic blood pressure the size of the groups is not large enough to enable us to arrive at such a sweeping conclusion. We look forward to the time when there will be enough material to solve this part of the problem.

### CONCLUSIONS

High blood pressure is a serious condition, more serious than most of us have hitherto thought it to be.

A high systolic or a high diastolic is apparently of more importance at the middle ages of life than at the younger or older ages.

The significance of pulse pressure seems generally to depend upon the significance of the systolic or the diastolic which accompanies it.

Dr. Hobbs—We will now hear from one whom we are always glad to hear on this subject of blood pressure and who has had a wide experience with the Northwestern in back of him. Dr. Fisher.

Dr. Fisher—Mr. President and Gentlemen: Mr. Hunter's and Doctor Rogers' paper is instructive and bears out the results obtained by the Northwestern on 4,165 rejected cases covering the years 1907-1920. The Northwestern furnished the Joint Blood Pressure Committee with 64,574 cases, issues of 1916-1920, carried to the anniversary of 1924. This data represents the blood pressure readings of members insured at standard rates and located in 87 localities. Examinations were made by well known, competent examiners. The mortality was computed by the American Men Table. The mortality on the entire block was found to be 10 points better than the general average mortality of the Company covering about the same period. This is no doubt due to the

examinations having been made by a selected list of examiners. The data was divided so as to give average proceeding by 5 millimeters, both below and above the normal average blood pressure for the age. In the groups below the normal average blood pressure group, there was but a slight difference from the mortality in the average group, but the ratio of the mortality to the "average" group was 9% higher in the group where the systolic pressure was 5 millimeters over the average for the age, and 59% higher in the group with an average systolic pressure of 10 millimeters over the "average" group, and 70% higher in a group showing a tension of 15 millimeters over the "average" group.

The same classification was made with respect to the (4th phase) diastolic. The lowest mortality was found in the class 5 millimeters below the normal or "average" blood pressure; in the class 5 millimeters above the normal, the ratio to the "average" group was 15% above, and in the class 10 millimeters over the normal, the ratio was 9%; while in the class 15 millimeters above normal, the ratio was 27%.

I hope to make use of our mortality cards for further study of the diastolic pressure. I am of the opinion that much can be learned by determining the mortality on the class:

- 1st. With a high systolic and an average diastolic;
- 2nd. A high systolic and a high diastolic;
- 3rd. A low or moderately low systolic and high diastolic.

The data of the Northwestern shows that a persistent (4th phase) diastolic of 90 mm. at ages under 40, and 95 mm. over age 40, marks the borderline. If the diastolic is taken at the 5th phase (or cessation of all sounds), the figures would be 95 mm. at ages under 40 and 100 mm. at ages over 40.

Attention is called to the table of average blood pressure

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at the different ages, compiled from the 64,574 cases, issues of 1916-1920, and also to the table composed of 20,735 cases, issues of October, 1922 to October, 1923; examinations made by the same class of examiners as in the data mentioned above. It will be noted that there is a decrease of from one to three millimeters in the later period, at ages 45 to 60.

Is it not possible for this Association to adopt a uniform table of average blood pressures? This would no doubt have a tendency to reduce blood pressure "shopping" on the part of life insurance solicitors.

The Northwestern data also demonstrates that a satisfactory class, with an average persistent systolic pressure more than 15 millimeters below the average for the age, can be selected.

There seems to be quite a large per cent of the medical profession who fail to realize the importance of hypertension in cases where no pathological changes are discovered to account for the same. What can we, as medical directors, do to correct this error and thereby prolong the lives of some of the members of our respective companies, as well as contribute, to some extent, to the welfare of the general public?

The conclusions to be drawn from the data presented by the New York Life and the Northwestern are:

That a persistent auscultatory systolic blood pressure of over 15 mm. above the average for the age, and a 4th phase diastolic of over 90 mm., at ages under 40, or 95 mm. at ages over 40, will show a high mortality.

### AVERAGE BLOOD PRESSURE

Average blood pressures for individual ages, based on 64,574 cases, examinations made by selected, competent Medical Examiners during the years 1916-1920.



TABLE I  
Average Blood Pressure

Age	Systolic	Diastolic	Pulse	Age	Systolic	Diastolic	Pulse
15	116 mm.	74 mm.	42 mm.	40	124 mm.	81 mm.	43 mm.
16	117	74	43	41	124	82	42
17	118	75	43	42	125	82	43
18	119	76	43	43	125	82	43
19	120	76	44	44	125	82	43
20	120	77	43	45	126	82	44
21	121	77	44	46	126	83	43
22	121	77	44	47	127	83	44
23	121	78	43	48	127	83	44
24	121	78	43	49	127	83	44
25	121	78	43	50	128	83	45
26	122	78	44	51	128	84	44
27	122	79	43	52	129	84	45
28	122	79	43	53	129	84	45
29	122	79	43	54	130	84	46
30	122	79	43	55	130	84	46
31	122	80	42	56	131	84	47
32	122	80	42	57	131	85	46
33	122	80	42	58	132	85	47
34	123	80	43	59	133	85	48
35	123	80	43	60	133	85	48
36	123	81	42	61	134	85	49
37	123	81	42	62	134	85	49
38	123	81	42	63	135	86	49
39	124	81	43	64	136	86	50
				65	136	86	50

The average blood pressures at individual ages were derived by a graphic process from crude averages by age groups for all years of issue combined, the average ages of the groups having been found respectively to be 16.52, 20.34, 25.15, 30.04, 34.93, 39.85, 44.83, 49.72, 54.64, 58.97, with a grand average age of 34.18. The crude averages referred to constitute a part of Table .

TABLE II  
Average Blood Pressure From 20,735 Cases, October, 1922, to October, 1923

Age	Palpatory		Auscultatory			
	Systolic	Systolic	Diastolic		Pulse	
20	116 mm.	120 mm.	4th Phase	5th Phase	4th Phase	5th Phase
25	117	121	78 mm.	73 mm.	42 mm.	47 mm.
30	118	122	79	74	42	47
35	119	123	80	75	42	47
40	120	124	81	76	42	47
45	121	125	81	76	43	48
50	122	126	82	77	43	48
55	124	128	83	78	43	48
60	126	130	84	79	44	49
			85	80	45	50

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Dr. Hobbs—I think these figures of Dr. Fisher will be very enlightening in guiding us if he will allow us to have them printed. Dr. Eakins.

Dr. Eakins—Mr. President and Gentlemen: In comparison with the diapason of the present duet sung by Rogers-Hunter any tone emanating from me would be the veriest peep. I had hoped my peep would be in harmony with their orotund offering, and would substantiate the truth formulated by Rogers years ago. You will remember his song, since repeated, that a small amount of data would be consonant, in essentials, with a more imposing array, if homogeneous. I am disappointed; really dismayed. Disappointed that my peep is so attenuated, through its paucity of figures, as to be almost completely muted. Dismayed that my expected harmony is discordant; that small and large data do not make a melody, and puts me in the position of a pupil disparaging one of the pet fundamentals of his master. My figures:

Issues From Jan. 1, 1925, to Sept. 1, 1926		
Number of of Cases	Expected Deaths by A. M. Select Table	Actual Deaths
97	.42	None

prove—if, indeed, they prove anything—that it is good to have a high blood pressure. Go thou and acquire a high blood pressure, if thou wouldst live!

Admitting my study of blood pressures is useless in this discussion, may it not be useful in another direction? Things which are equal to the same thing are equal to each other. No one in his right mind will dispute the fallacy, indicated by these figures, of the conclusion that a high blood pressure is good to have. Other figures of which we have heard, indicate that a better mortality is experienced in so-called non-medical business than on examined business. If the conclusion from these figures is, as it is, absurd, may not the rules of logic be employed on a comparison of it, with a like

conclusion concerning the mortality from non-medical business? The question is raised, with full knowledge that a logical conclusion must be based upon sound premises. I have as much enjoyment laughing at myself, as at anything else. You will, then, gather that my risibility is easily stirred, but never yet have I been able to summon even a smile, unless it be a derisive one, at the possibilities of an exceptionally favorable mortality from either high blood pressures or unexamined business.

In order to indulge in something other than just words, other than only *risus sardonicus*, I might offer a crumb which may be of use to us if we can properly interpret it. Recent studies on the pathogenesis of hypertension, conducted by Bordley and Baker, Johns Hopkins Pathological Department point to the presence of an arteriosclerosis in the medulla oblongata. They have always found an arteriosclerosis in the medulla, in all cases with a history of hypertension; and did not find it in cases without that history. If our physiologists will interpret that fact for us, in terms of a working hypothesis, then my ululation will not have been in vain.

Dr. Hobbs—Gentlemen, does any one else want to say anything on the subject of blood pressure? If not, I will call on Dr. Frost.

Dr. Frost—I have a point of view on blood pressure which probably is a combination of the clinical and the life insurance and is based entirely upon my own study of New England Mutual material.

In the first place, I think it is perfectly all right for us to insure our applicants on the basis of mortality figures worked out, so many millimeters above and below. Apparently we are stopping right there. The only way we are going to get any farther is for us to utilize our material to the full. We can't do that without correlating it with clinical knowledge. In my estimation of hypertension, so-called

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hypertension, at the middle ages, it does not exist. I believe before that, in the earlier years of life, you have transient hypertension, the cause of which I do not know, but we all know perfectly well that we get a large number of younger individuals who, under the stress of excitement and unusual exertion, will show a transient hypertension. Studying the figures still farther, we notice that with increasing age, these hypertensions can become permanent. Whereas at first in the earlier ages, you have a systolic hypertension and practically no diastolic, you gradually get a rise in the diastolic and in the later decades of life, you have the sustained hypertension with a high systolic level permanent and a high diastolic level permanent.

Now, it is probably a recognizable fact that 75 per cent of cases of heart diseases are due essentially to hypertension in the beginning and that the cause of that disease is hypertension. Arteriosclerosis develops into heart disease and then probably branches out in several directions, either along the cerebral route with apoplexy, thrombosis, etc., or continuing along the cardiac group with cardiac failure and branching off with uremia or along the diabetic route, so that I would not agree with Dr. Rogers' observations that the early hypertensions are negligible. I believe that if we are able to follow these individuals with hypertension at the earlier ages long enough, that type which we call the excitable individual and now considered more important, if we follow him long enough, we are going to find that he is the potential hypertension case, the potential circulatory disease case, and I believe that the decade from 40 to 50 is the critical decade for circulatory diseases. Why would not that be an explanation of the increased mortality of entrants during that decade?

Dr. Rogers—I should like to reply to one phase of Dr. Frost's remarks and that is with regard to the hypertension at middle life, or that condition we ordinarily speak of as

essential hypertension, it being the descendant of the slight hypertension of earlier life. Summing up our two groups of statistics, we have a continuous story to the effect that the mortality is very low under age 40, however long you watch them. Those are entrants below age 40, persons aged under 40, when they enter. Although we keep them under observation a long time, they don't show a high mortality, but where we get a hypertension in the middle period of life, between 40 and 50, the mortality is very high. I don't know, but I suppose that means a study of twenty years on the whole, certainly over fifteen years, and it does not show a high mortality among those in whom a high blood pressure is found in the early period of life, however long they are kept under observation.

I have been very much interested in the discussion of this subject and have very little further to say. I haven't much sympathy with Dr. Fisher's advocacy of the fourth phase. The consensus of opinion among the Medical Directors seems to be that the fifth phase is more easily understood and it is certainly much more generally used. I doubt whether it is worth while to be very meticulous about whether it is the fourth phase or the fifth. All we are anxious to know is whether the blood pressure is high or not high. All that we can say positively is that cases in which the blood pressure is clearly high, that is, more than 25 mm. above the average, have an extremely high mortality about the middle period of life. I believe that this is all we know about the subject at present and it is hardly likely that we shall know much more about it for a number of years to come.

Dr. Fisher—Dr. Rogers misunderstood me. I am not advocating but you don't know what phase you have got.

Dr. Rogers—I don't care in any given case whether it is the fourth phase or the fifth. We can draw inferences

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enough whichever it is. That is the point I wanted to make.

Dr. Hobbs—Gentlemen, before luncheon is served, I want to give a few minutes to Dr. Cragin, who has a subject he wants to present to you and after that we will have luncheon. At 2:00 o'clock, I want everybody back here promptly, because we have a very important program we want to get through with this afternoon. Dr. Cragin.

Dr. Cragin—Mr. President and Gentlemen: With the development of the Folin-Benedict test for glycosuria it immediately became necessary to depend on a colorimeter for comparison. After trying the various colorimeters of the liquid sort it was felt that they failed in several particulars:

In the first place, there was some question about the constancy of the color.

In the second place, no one combination of chemicals had been approved as standard for making up these mixtures.

In the third place, the handling of the tubes containing the liquids led to breakage. They were hard to keep clean, and finally we found that the varying backgrounds of sky, cloud, buildings, and degree of sunlight were very confusing in obtaining an exact comparison.

After some experimentation in various fields, notably with aniline dyes, we were convinced that the only non-fadable color which we could obtain would be that of glass. We, therefore, began some experiments with colored glass and finally were able to obtain from the Corning Glass Works a standard set of colors representing the different percentages of sugar as checked up through the Folin-Benedict test with dextrose.

The question of lighting was easily solved by the Macbeth light which needs no introduction as it is well known that the light emitted from the Macbeth apparatus is identically the same spectroscopically as that of ordinary daylight.

It only remained to make the combination of these factors

which we now present to you. A few of the features of this apparatus are as follows:

1. The background of light is constant no matter what the climatic conditions may be. This apparatus can be used in the darkest corner of the building or right up against the window without any difficulty.

2. The glass furnishing the colorimeter is constant. These have been carefully worked out with the assistance of Dr. H. P. Gage of the Corning Glass Works, and we feel that the colors are as near an absolute match as is possible to obtain.

3. There is no soiling of the colorimeter. We have found by careful examination and analysis that an aperture of the size represented in the colorimeter as shown, namely 11 cc. by 45 cc. gives the proper visage for comparison, doing away with the interference of the narrower portion of the liquid at the side of the test tube.

As a by-word I may say right here that one of our principal difficulties has been in getting tubes of a constant caliber, but I am assured by Dr. H. P. Gage that any time that we fix upon the proper standard of tube that they can be furnished in the pyrex glass in any quantity from the Corning Glass Works.

Another feature of the colorimeter is that the front is easily removed for the cleansing of the glasses. If, by any violent accident, the glasses should become injured, they can be slipped out and replaced. The standard is adjustable and the apparatus may be used with or without the Macbeth light.

I present this apparatus to you for your comment and criticism.

Dr. Hobbs—Dr. Cragin has the apparatus outside and I hope you will all take the opportunity of seeing it. We have just a few minutes to spare before luncheon and I want to take this opportunity of introducing my successor, Dr.

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Beckett, who will occupy the chair for the rest of the afternoon. I have served this association in an official capacity for ten years. It has been a pleasure to serve you and I am glad to have had the opportunity of doing official work for you for the past ten years.

Yesterday I received this gavel from Dr. Beckett, made of red-wood from California. I appreciate it very much and shall keep it as a reminder of my associations with this organization.

Now, I will ask Dr. Beckett to step up here and say a few words to you for a moment. Dr. Beckett.

Dr. Beckett—Mr. President and Members of the Association: It is not for me to make a speech at this time. I am afraid, if I should, I would be something like the young man who wanted to be an orator. He wanted to so badly that he prepared a lecture, advertised it and rented a hall. In the afternoon of the day that he was going to give the lecture, he went down to the box office to see how the tickets were selling and as he went down, he met a little girl with a little dog running along by her side and he said to the little girl, "Are you coming to the lecture tonight?" She said, "No, we are not coming." "Why are you not coming to the lecture," he asked. "Well," she said, "we haven't got enough money to pay for the tickets. I would be glad to give you the dog, but we haven't got the money." He was so taken with her that he said, "Come on down to the box office and I will give you enough tickets for you and your folks." During the lecture, he looked down into the audience and saw the little girl with her folks. As he went out he met her and said, "I see you all came to the lecture tonight. How did you like it?" "Well," she replied, "I'm glad I didn't give you the dog."

I want to thank you for this very great honor that you



have conferred upon me. I am sure it is not merited and it comes to me with very much more feeling than it would, otherwise, as it is probably your friendship that has prompted this action on your part rather than what I have done for this Society to merit the position. I also want to thank you for the compliment extended to the company that I represent. I have been asked by a number if the meeting was going to be out on the coast next year. I said, "I have nothing to do with the selection of the place of the meeting." I am quite confident that the committee that has that in charge will select this city, which, of course, will be agreeable to me. I would like very much to see you come, if it were the disposition of the Society and the committee to meet in Los Angeles. I will promise you a good time if you come and it would delight not only me but my company to entertain you at that time and we would probably do it as my father used to say, "We would detain you in a hospital manner." And I don't know but what it would be a good thing for some of you who have become oblivious to the fact for so long that you are near Hell Gate that it gives you no concern whatever, I think that it would be refreshing to get out and see the Golden Gate. You might not all pass through it, but it would be refreshing to see it.

So I will leave this matter to you and I want to again thank you for the great compliment and the great honor you have conferred upon me. I feel that after this splendid meeting, presided over so splendidly by the retiring President, that I have a very hard position to fill and it will only be by your hearty support that we can begin to come up to the standard that has been set me by my predecessor. I am sure that you will give me the support that you can. Thank you.

A motion was made by Dr. Toulmin that the Association extend a vote of thanks to the New York Life Insurance

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Company for its hospitality during the two days of the convention. The motion was seconded and carried unanimously.

### AFTERNOON SESSION

Dr. Hobbs—I believe we have a very important part of our program ahead of us, and while we wish to take all the time necessary, I am going to ask those who will participate to be as quick as possible and get in their thoughts from themselves to us. We don't want to curtail the time anybody has but at the same time I feel as though I would be justified in cutting the remarks short unless they are all to the point.

The next on the program will be "Mitral Insufficiency—a Limited Experience, Including Etiology" by Dr. Frank L. Grosvenor.

## MITRAL INSUFFICIENCY—A LIMITED EXPERIENCE, INCLUDING ETIOLOGY.

BY F. L. GROSVENOR, M. D.

*Medical Director, The Travelers Insurance Company*

The inclusion of "Etiology" in the title of this paper is predicated on the assumption that certain infections have an etiological bearing on the development of heart disease. The experience is necessarily limited. Six thousand, eight hundred and fifty-eight lives were studied, as per the tables below, which are submitted for your consideration.

The exposure is on the policy year basis and in other respects is comparable with experiences given in the American-Canadian Mortality Investigation.

Circulatory Diseases, as a cause of death, were also compared with that of the American Men Table in each group. Included as Circulatory Diseases were cerebral hemorrhage and apoplexy, pericarditis and acute endocarditis, organic disease of the heart, angina pectoris and diseases of the arteries.

An extremely interesting point to me is the general trend indicating a higher mortality at the younger ages of entry.

TABLE NO. I  
Mitral Insufficiency—Without Hypertrophy  
(Substandard Policies)  
Years of Issue 1905-1924—Exposure 1905-1925

Age at Entry	Exposure	Actual Deaths	American Men Expected Deaths	% Actual to Expected
		First Five Years		
— - 29	7,117	47	26.4	178.2%
30 - 44	8,551	65	39.5	164.4
45 - —	745	8	6.4	125.0
Total	16,413	120	72.3	166.0
		Sixth and Subsequent Years		
Att. Age				
20 - 34	1,823	13	8.1	160.4%
35 - 49	3,432	44	22.7	193.8
50 - —	568	12	8.5	141.8
Total	5,823	69	39.3	175.7

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### All Ages at Entry Combined

Ins. Yr.				
1	4,284	34	14.0	243.1%
2	3,648	12	16.0	74.9
3	3,267	37	15.4	240.1
4	2,821	19	14.0	135.8
5	2,393	18	12.9	139.4
Ult.	5,823	69	39.3	175.7
Total	22,236	189	111.6	169.4

In this group, the expected deaths from Circulatory Diseases were 10.5, the actual 81.

TABLE NO. II

### Mitral Insufficiency—With Hypertrophy (Substandard Policies) Years of Issue 1907-1924—Exposure 1907-1925

Age at Entry	Exposure	Actual Deaths	American Men Expected Deaths	% Actual to Expected
First Five Years				
— - 29	2,105	17	7.8	217.5%
30 - 44	2,778	30	13.0	231.1
45 - —	259	1	2.2	45.6
Total	5,142	48	23.0	208.7

### Sixth and Subsequent Years

Att. Age				
— - 34	611	6	2.7	221.3%
35 - 49	1,313	10	8.7	114.2
50 - —	230	3	3.5	86.6
Total	2,154	19	14.9	127.2

### All Ages at Entry Combined

Ins. Yr.				
1	1,299	8	4.3	186.8%
2	1,123	11	5.0	220.0
3	1,023	16	4.8	329.3
4	897	3	4.5	66.8
5	800	10	4.4	229.2
Ult.	2,154	19	14.9	127.2
Total	7,296	67	37.9	176.6

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Here the expected deaths from Circulatory Diseases were 5, the actual 33.

Comparing Tables Nos. I and II, it will be seen that the mortality in cases with hypertrophy is not much over that in cases without hypertrophy, differing in this particular with the experience given us by Dr. Rogers. The limited experience and difference in selection may account for this.

TABLE NO. III  
Hypertrophy—Without Heart Murmur  
(Substandard Policies)  
Years of Issue 1907-1924—Exposure 1907-1925

Age at Entry	Exposure	Actual Deaths	American Men Expected Deaths	% Actual to Expected
First Five Years				
— - 29	239	1	.9	113.5%
30 - 44	261	3	1.1	260.9
45 - —	54	—	.6	0
Total	* 554	4	2.6	153.2
Sixth and Subsequent Years				
Att. Age	67	—	.3	0%
— - 34	103	—	.7	0
35 - 49	28	1	.5	214.1
50 - —	—	—	—	—
Total	198	1	1.5	68.2
All Ages at Entry Combined				
Ins. Yr.	154	—	.5	0%
1	128	—	.6	0
2	108	2	.6	354.6
3	91	1	.5	199.2
4	73	1	.4	245.1
5	198	1	1.5	68.2
Ult.	—	—	—	—
Total	752	5	4.1	122.6

The expected deaths from Circulatory diseases in this group were 4, the actual 2.

The mortality is less than I anticipated. The exposure is probably too small to permit the experience to be of much value.

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TABLE NO. IV  
Mitral Insufficiency—With Rheumatism, Tonsillitis and Streptococcic Infection  
(Substandard Policies)  
Years of Issue 1907-1924—Exposure 1907-1925

Age at Entry	Exposure	Actual Deaths First Five Years	American Men Expected Deaths	% Actual to Expected
15 - 29	2,216	20	8.2	245.1%
30 - 44	2,208	21	10.2	206.7
45 - —	157	—	1.3	—
Total	4,581	41	19.7	208.6
Sixth and Subsequent Years				
Att. Age				
20 - 34	603	8	2.7	298.6%
35 - 49	1,078	12	7.0	171.8
50 - —	176	5	2.5	194.8
Total	1,857	25	12.2	204.4
All Ages at Entry Combined				
Ins. Yr.				
1	1,216	9	3.9	231.9%
2	1,019	3	4.4	68.5
3	914	16	4.2	379.5
4	777	7	3.8	186.5
5	655	6	3.4	175.3
Ult.	1,857	25	12.2	204.4
Total	6,438	66	31.9	207.0

Fifty-four per cent of this group gave a history of rheumatism, while forty-six per cent gave a history of streptococcic or perhaps allied infections such as tonsillitis, scarlet fever, erysipelas and sore throat.

The expected deaths from Circulatory Diseases were 3.9, the actual 39.

TABLE NO. V  
Mitral Insufficiency—With Acute Infection Not Streptococcic  
(Substandard Policies)  
Years of Issue 1907-1924—Exposure 1907-1925

Age at Entry	Exposure	Actual Deaths First Five Years	American Men Expected Deaths	% Actual to Expected
15 - 29	1,401	16	5.2	307.6%
30 - 44	1,761	23	8.0	288.2
45 - —	249	2	2.2	90.8
Total	3,411	41	15.4	266.5

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Sixth and Subsequent Years				
Att. Age				
20 - 34	330	1	1.5	68.1%
35 - 49	732	6	4.8	123.8
50 —	141	1	2.1	47.1
Total	1,203	8	8.4	94.8
All Ages at Entry Combined				
Ins. Yr.				
1	905	9	3.0	300.4%
2	761	3	3.4	88.1
3	676	12	3.3	367.8
4	587	7	3.0	231.5
5	482	10	2.7	370.9
Ult.	1,203	8	8.4	94.8
Total	4,614	49	23.8	205.7

The expected deaths from Circulatory Diseases in this group were 3, the actual 11.

Considered as non-streptococcic infections were typhoid fever, diphtheria, influenza, appendicitis, diseases of the gall bladder, pneumonia, empyema, carbuncles, mastoiditis and otorrhoea, chronic ulcers and diseases of the teeth and gums.

There is less difference in the mortality between Tables IV and V than I had anticipated. It will be noted that the ratios in the select experience in Table V are considerably greater than in Table IV, which is very likely accidental, although the deaths from Circulatory Diseases give a much higher ratio in Table IV than in Table V. Tables IV and V show a mortality considerably in excess of Tables I and II, which include both Groups IV and V, as well as the cases in which there was no history of infection. A separate classification of cases without a history of infection was not made for the reason that a negative history in Life Insurance examination is no criterion of a negative history in the individual.

Dr. Hobbs—We will hear from Dr. Cook.

Dr. Cook—Mr. President and Gentlemen: Dr. Grosvenor's paper brings before us what I believe is the most interesting and important subject at present before the inter-

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nist, as well as the medical director, *i. e.*, the interpretation and evaluation of systolic apical murmurs. Any controversial subject is interesting, but especially one of which we had felt so comparatively sure in our conceptions and where these conceptions are so frankly challenged. I know of no diagnosis of which we have probably felt more certain than that of an applicant with a history of acute articular rheumatism, a loud systolic murmur at apex transmitted to axilla or back, accompanied by displaced apex, apparent hypertrophy, and accentuated second sound.

As examiners or physicians we have emphatically pronounced the case one of mitral regurgitation, and have explained how the valves had been damaged so that they leaked blood back into the auricle during systole, and we have felt thoroughly modern if we further explained that a few years ago such a man would have been told he had only a short time to live, whereas now we know such a lesion is compatible, under proper care, with a long useful life.

As medical directors we have classified the case as definitely mitral regurgitation, and declined or assessed 175 to 200 per cent rating, according to the policy of our company in considering impaired risks. Now we are emphatically told by a careful pathologist, internist, and professor of medicine at Harvard, that such a case is never mitral insufficiency, that it may be mitral stenosis, but more likely the heart is entirely normal. To use Cabot's own words, "Granting that mitral insufficiency exists as a great rarity, it is not a clinical entity, for it cannot be diagnosed during life."

Two scientific statements could scarcely be more contradictory. Cabot is not unsupported in this position. Dr. Paul White, also of Boston, takes a somewhat similar position, though not so extreme, and a review of recent literature shows that Mackenzie, Lewis, Vaquez, and other cardiologists have somewhat similar opinions, or at least they are alike



in opposition to our classical conception of mitral insufficiencies and the significance of systolic murmurs.

While it is true that from the life insurance viewpoint the essential fact is that these policyholders with systolic murmurs at the apex have shown 175 to 200+ per cent mortality, nevertheless, we cannot wisely ignore Dr. Cabot's demand for a more discriminating position than we are now taking, unless we can successfully controvert the convincing pathological and clinical "facts" detailed in his book. On the other hand, if policyholders with a systolic murmur at the apex live six to eight years less than policyholders without the murmur, it is certainly as important a "fact" as anything brought out in Dr. Cabot's book, and the statistics furnished by Dr. Rogers, Dr. Grosvenor, and others, seem to offer testimony which Dr. Cabot cannot wave aside as an "outworn medical legend"—as he terms our conception of the significance of systolic murmurs. These applicants may not have heart disease, as some cardiologists assert, but nevertheless they do not live as long as applicants without the murmur, and a considerable percentage of them die of what is certified to as heart disease.

It is an interesting problem as to what should be the position of life insurance medicine towards these murmurs, in the light of such conflicting conceptions. It seems to me the first step has already been taken by the M. I. B. coding committee in substituting physical findings in our code for attempted diagnosis. In the present confused state of our knowledge of cardiology, I think it can be positively stated that the usual insurance examination does not permit of a diagnostic interpretation of these murmurs, and we are only clouding the issue and blocking a better understanding of the true condition by continuing the old classification. I believe we must learn to think and write in terms of the M. I. B. heart murmur code, or, in other words, in actual findings in-

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stead of snapshot diagnosis. This code is admittedly tentative and will require modification as our experience grows, but for our purposes it is the best classification so far offered.

The etiological classification urged by Fahr and others, in view of the cursory history and examination in an insurance examination is not practical: for example, a Wassermann is only infrequently available. The structural classification, *i. e.*, myocardial, endocardial, pericardial, size, position, etc., is what we have previously attempted and by which we have been led so far astray. The functional classification of certain heart conditions, *i. e.*, heart failure, extent to which normal functional activity can be maintained, disordered heart action, irregularities, etc., has been attempted to a very limited extent in life insurance medicine. For example, we have classified tachycardiacs, bradycardiacs, and extrasystoles; a functional exercise test has been used rather extensively, such as described today by Dr. Patton; and vital capacity tests have been tried, but I believe dropped into disuse; and more recently an elaborate blood pressure test has been offered by the New England Mutual. Many eminent cardiologists are, however, emphatic in criticizing any exercise or forced respiratory or blood pressure tests.

I believe that a reasonably large experience under our new code classification of physical findings will give some very valuable light on the proper interpretation of heart murmurs, which will not only permit a more accurate evaluation to insurance medicine, but will materially assist the conceptions of clinical medicine. At the present time we may watch interestedly the controversy as to the pathological interpretation of murmurs, but with the very definite excess mortality experienced by insurance companies with systolic heart murmurs, we must continue to rate them for a mortality varying up to 200+ per cent of the expected, no matter whether the true diagnosis is mitral insufficiency, mitral stenosis, effort

syndrome, or normal heart. These people show an excess mortality and they die of heart disease, according to the death certificates, and, unfortunately as policyholders, the financial loss to the insurance companies is just as great whether or not the optimism of the modern cardiologist reassures them as patients in regard to the insignificance of the finding.

I always remember old Dr. Lambert's comment thirty or more years ago about albuminurias. There was then the same controversy as to whether albuminuria meant renal disease or might be purely "functional." He pointed out that no matter whether it was organic or functional, the albuminurias accepted by the Equitable died in a greater proportion than policyholders without albuminuria. And I also remember a remark of Dr. Rogers' that when he accepted an applicant with a murmur on the opinion of an especially prominent internist that the condition was functional, an early death loss might be expected. Medical opinion proverbially swings to extremes, and the more prominent the essayist, the more extreme is apt to be his position. This is natural—these men have the inspiration of genius, they see the fallacy of a prevailing opinion, and they inevitably become propagandists. As Sprague and White state it, "Fashions in medical diagnosis are so variable, and beliefs of different men so actively defended, that it is difficult to take a middle course. Most surely is this true in the diagnosis of organic mitral insufficiency."

Another reason, I believe, why there is such a marked discrepancy between the conceptions of the cardiologist and the medical director is that they see entirely different types of cases. The medical director is considering apparently normal active individuals in professed good health. The clinician sees the complaining, the ill, the moribund.

Comparing the clinical notes of auscultatory findings in a

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moribund laborer with the necropsy findings a few days afterwards will throw valuable light on many questions, but it may seriously mislead us if we apply the interpretation too literally to the active banker who applies to us for \$100,000 of insurance, and who does not admit a day's illness in his life.

Dr. Grosvenor's and other life insurance figures are convincing to me that a company will suffer loss, which will accept these systolic murmurs freely at standard rates on Dr. Cabot's assurance that mitral regurgitation does not exist, and Dr. Mackenzie's belief that no one ever dies of it.

While we may be justified, as Dr. Rogers has recommended, and as I imagine most of us are doing in practice, in accepting some faint or soft systolic murmurs at the younger ages at standard rates, and can perhaps gradually, but carefully enlarge the group of functional diagnosis, nevertheless, the class of applicants which we now perhaps improperly call examples of mitral insufficiency, corresponding to the classical picture of rheumatic history, systolic murmur and apparent hypertrophy, will, as far as we are now able to judge, experience a decreased longevity—and classified by age and degree of hypertrophy, will show a mortality closely corresponding to Dr. Grosvenor's experience. Until the time comes when the practical limits of an insurance examination permit us to interpret our finding more accurately in terms of longevity, I believe we will do well not to modify our present position very greatly.

Our interest in heart disease is emphasized by the increasing importance of the lesion, both from an insurance and public health point of view. Dr. Dublin has recently estimated the deaths in the United States from heart disease at over 200,000 annually, and he believes the pre-eminence of the disease will increase as a cause of death. He estimates that over 2,000,000 in the United States are now suffering

from heart disease, and the majority of these are at the age when the larger amounts of insurance are applied for.

I should like to suggest that we appoint a committee to prepare a brief statement giving some of the newer viewpoints in heart examination, diagnosis and prognosis, which we could send to our examiners. There would be a great advantage in having a committee prepare such a statement, which any company that desired could use, as it would tend to give all examiners a similar understanding of what is desired in a life insurance examination, and tend to give our resulting statistics greater value.

We should certainly stress symptoms more than we have, especially endeavoring to bring out the presence of dyspnoea or discomfort or pain in the chest on exertion, and not limit our attention to physical findings. Mackenzie has been the pioneer in urging the importance of symptoms. Cabot shows that dyspnoea was the earliest symptom in 53 out of 61 fatal heart cases, and 60 out of 80 out-patient cases.

We should urge careful attention to the presence of a diastolic murmur in all cases where a systolic is heard at the apex. Cabot and others claim that we are missing diastolic murmurs in cases of mitral stenosis, and so wrongly calling them insufficiencies.

We are planning the installation of an X-ray equipment at our Home Office, and I believe this will become more common, as it is certainly the only sure way to determine the important point of size and position of the auricles and ventricles. We have been making more and more use of the X-ray in doubtful cases, and have derived a great deal of help. Arrangements can easily be made in cities and many larger towns for a competent X-ray opinion on the heart and aorta, for a small fee—usually \$5, and I believe the committee above mentioned, if appointed, could arrange to furnish our companies with a list of members of the American

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Roentgenological Society, and their addresses, who would make such an examination for that fee. In my opinion, \$5 for a Roentgenological report on heart and aorta in any case of \$10,000 or more, at age 50 and over, would be a lucrative investment.

In closing, I want to express my appreciation of Dr. Grosvenor's interesting and convincing presentation of the excess mortality to be expected in cases of systolic apical murmurs.

Dr. Hobbs—I think that a great many endorse what Dr. Cook has said in regard to the work of Dr. Cabot and also in regard to the publication of Dr. MacKenzie, now deceased. I am quite sure that we can follow these gentlemen in our selection, taking their ideas as to the seriousness of the impairment of mitral insufficiency. It may be that we can separate the sheep from the goats and make a classification of mitral insufficiency where we could be liberal in one part of the business and not so liberal in other parts of the business. That will be for you to decide. I wish Dr. Cook would put the name of his committee and his object in writing and let me have it and we will see about appointing such a committee to report in the future.

We will now return to the discussion of the subject. Dr. Frost.

Dr. Frost—Mr. President and Gentlemen: The title of Dr. Grosvenor's paper, "Mitral Insufficiency," reveals to us a field of intriguing interest. It is a diagnostic label of which, if we are to be as accurate as modern clinical evidence will permit, we must be rather shy.

A score of years ago it presented no particular problem. Given a systolic murmur at the apex, of sufficient intensity to be transmitted to the left axilla and back, with accentuation of the pulmonic second sound and with enlargement of the heart evidenced by inspection palpation and percussion,

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the diagnosis was not to be questioned. Of course it was pure mitral regurgitation, due either to inflammatory valvular disease or to dilation of the valvular orifice.

With the passage of the years, however, we are becoming less certain of our ground, less placid in our deductions from these three classical signs. At this day apparently we can never be sure of the condition of the mitral valve; neither from the clinical signs, nor from any other evidence which may be at our disposal.

We were given an inkling of this nearly 27 years ago, by Dr. Jerome Kingsbury, in a short statistical study entitled, "Heart Murmurs and Heart Lesions," published in the "Surgical and Medical Report of the Presbyterian Hospital," January, 1900. Dr. Kingsbury designed his study "to show the relation existing between endocardial murmurs and the condition of the valves as found at autopsy." His material was obtained from the records of 720 autopsies made at the Presbyterian Hospital. Of these, 379 (52+) revealed hearts with normal valves, with no history of murmurs. In 138 (19+) valvular lesions were found which had apparently produced murmurs. In 83 (11+) valvular lesions were found without a history of murmurs; of which lesions, 53 (63+) involved the mitral valve, either alone or in combination with other valves. In 120 (16+) valvular lesions were not found in spite of a history of murmurs. Of these, 34 (28+) were located at the apex.

Very recently Dr. Richard C. Cabot has published in his book "Facts on the Heart" an imposing mass of evidence, based on the study of 1906 cases of heart disease, which tends to destroy the old ideas—he calls them "legends"—about mitral regurgitation. The change in his point of view, as shown by a comparison of his earlier and recent publications, indicates very well the trend of authoritative opinion. In 1909, in the fourth edition of his "Physical Diagnosis,"

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he was positive in his description of the signs of mitral regurgitation. In 1926, he is astonished "how long the legends about mitral regurgitation have persisted and on what good authority they have rested." To quote him farther, "But granting that mitral regurgitation probably exists as a great rarity, it is not a clinical entity, for it cannot, so far as I see, be recognized in life. The classical trio of signs \* \* \* which have been supposed to point to mitral regurgitation, have occurred many times in this series without any evidence post mortem that the mitral valve was diseased or incompetent." "The old point of differentiation between functional and organic murmurs heard best at the cardiac apex, namely the transmission of the supposedly organic murmurs to the left axilla and back, has been proved to my satisfaction to be false. I have heard many such murmurs which showed nothing particular to account for them at necropsy, not even cardiac enlargement, no increase of the valve circumference, and no disease of the valve itself." He continues, "In life insurance examinations this certainly is a point of real importance. It is a serious thing to be refused life insurance because of the false interpretation of a systolic murmur." With this latter statement we are in hearty accord.

This conception of the impossibility of deducing definite valvular lesions from heart murmurs is not a stranger to life insurance medicine. During my five years of association with the New England Mutual Life Insurance Company I have heard Dr. Dwight repeatedly assert his conviction, based upon his own experience and observation, that no clinician, however great his ability, could consistently diagnose valvular disease by means of cardiac signs.

As it therefore appears that we cannot safely presuppose a mitral valve lesion from any combination of cardiac signs, I am assuming that Dr. Grosvenor, in using the term, "Mitral Insufficiency," has in mind a systolic murmur at the apex,



constant rather than intermittent, localized or transmitted to the left, with no insistence as to an underlying heart lesion. It is obvious from his statistics, as from those of Rogers and Hunter presented in their "Mortality Study of Impaired Lives—No. 3," that apical systolic murmurs, whether clinically of the apparently nonorganic type, whether associated with enlargement of the heart or with a history of infection, carry with them a definitely increased mortality; less marked in the nonorganic type, more marked in the presence of enlargement, most marked when combined with a history of infection as a possible etiological factor.

It must also be true that in a large group of these impairments there are subgroups which, if we could determine them by increased skill in selection, would afford much lower mortalities. Some of them would undoubtedly be eligible for standard insurance. This is the problem with which we are at present struggling in the Medical Department of the New England Mutual Life Insurance Company.

Our material in this particular group of apical systolic murmurs is not as yet sufficient in volume or length of exposure to warrant a study of mortality. I propose to outline our method of procedure and the reasons upon which it is based. Inasmuch as the cardio-respiratory test is intimately concerned with it, I shall conclude with a brief statement of our experience with it.

As a preliminary consideration, we assume that no constant apical systolic murmur, however harmless it may appear from the clinical signs, may safely be considered innocent from the life insurance point of view. Furthermore, we do not believe that any accurate conception of the condition of the mitral valve or myocardium may be derived from analysis of clinical signs. As a corollary, we feel that a satisfactory attempt to select the favorable subgroups must be based upon mortalities derived in the first place from analysis of

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carefully recorded signs and symptoms, and in the second place from analysis of accurate observations of the behaviour of the cardio-vascular apparatus when subjected to strain.

We believe that a seriously damaged heart, when subjected to strain, will give some hint of such damage by reason of abnormality in the nature of the reaction. Therefore we apply a strain by means of the cardio-respiratory test.

It appears reasonable to suppose that a heart with an apical systolic murmur which arises from actual damage will, if this damage is severe enough to interfere with cardiac action and cause increased strain, gradually accommodate itself to do an increased amount of work. In other words, it will hypertrophy. We, therefore, take into consideration the duration of the murmur. Frequently, of course, there is no satisfactory evidence as to this. However, if it can be traced back for five or more years and the heart does not appear enlarged, we incline to the opinion that the murmur cannot arise from any serious cardiac lesion.

We are influenced to a considerable extent by the physical characteristics of the murmur. The intermittent, cardio-respiratory type, which disappears at certain stages of the respiratory cycle, we think to be of no serious import and in the absence of disturbing concomitant signs largely disregard it. The intermittent murmur which is apparent only in the recumbent position, disappearing in the upright, we feel to be of benign nature. However we are not quite so sure of this as in the preceding type and consequently check it with the test. If the reaction is satisfactory we disregard the murmur. The harsher murmurs, which by reason of their volume are transmitted over the heart area or outward to the left axilla or back, we consider more seriously and handle somewhat more rigorously. This may not be justified from clinical evidence, but we think it would be foolhardy to dep-

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recate their importance until we have learned more of their potentialities.

Upon the accentuation of a murmur following exercise, or the elicitation of an evanescent systolic murmur by exercise, we place but little significance. Exercise naturally increases the force and rate of the heart contractions. It is only to be expected that an existing murmur will be accentuated, and that sound vibrations ordinarily inaudible will be temporarily increased to the range of audibility. It is difficult to see how this phenomenon offers us any real information.

We believe a good deal of information may be obtained from the character of the heart sounds. In a thick-chested individual, muffling of the apical sounds is to be expected. Provided the first and second sounds are equally subdued, we are not worried. However, if either of these sounds is subdued in relation to the other, we believe an impaired heart should be suspected. A reduplicated pulmonic second sound we think to be of rather serious import, as clinical evidence indicates that mitral valvular damage is rather apt to be present.

In addition, of course, we give particular attention to such details as the rhythm and force of the heart contractions, blood pressure and condition of the peripheral circulation; and the history of infection or circulatory symptoms. We feel that we obtain somewhat more accurate information as to these points than would be possible in the ordinary examination, in that every one of these cases is examined with care by one of our chief examiners: men selected for such work on account of their outstanding ability. Finally, we check the condition of the kidneys by a complete chemical and microscopic analysis of the urine, which must conform to a rather rigid standard.

All this information may be acquired from a careful examination. It is directed entirely toward the endeavor to

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determine whether there is any damage to the cardio-vascular or renal systems, and if so, it's extent.

As a final check we apply the cardio-respiratory test. If the murmur is the sole impairment and the reaction to the test is good, we are inclined to recommend regular policies. If the reaction is poor, we decline the risk. In the presence of other impairments suggesting a damaged heart, if such damage does not appear excessive or progressive, with a normal test, we are willing to consider risks at the younger ages, roughly under age 35, usually for endowments or non-renewable and nonconvertible five-year terms. Such risks over the age of forty we do not as a rule care to consider, except for very short endowments which are usually financially prohibitive for the applicant. It is not possible in the scope of this discussion to go into detail. Suffice it to say that with an abnormal response to the test, we decline. With a normal response, we may decline because of the serious aspect of the clinical picture as a whole; we may recommend regular policies, or limitation to forms having greater reserves and earlier maturities.

It is of interest, at least to us, that a large proportion of the applicants of this class whom we have declined have given an insufficient or "hypoactive" reaction to the test, evincing mainly an inability to raise the systolic pressure to a satisfactory level in Steps 6, 7 and 8. We have already noted this peculiarity in certain cases of apparent mitral stenosis. We are wondering whether some of these may not have mitral stenosis. This would be in accord, I believe, with clinical evidence as to the frequency of mitral stenosis, in the presence of apical systolic murmurs and without demonstrable pre-systolic murmurs.

Our experience with the cardio-respiratory test has been very satisfactory so far. It is now nearly five years since we developed it. Up to September, 1926, we had submitted to

it 3839 applicants. We accepted 2498 of these, 65.07%. The report of our actuary, Mr. Herbert B. Dow, as to the mortality investigation of the policies issued follows:

"This investigation included 2557 policies with insurance of \$18,402,002. Excluding cancelled and reissues, there remained 2135 policies for insurance of \$15,343,500. The investigation was closed in September, 1926, with a maximum duration of five years for any insurance. The percentage of deaths by the American Experience Table was 13.12 of the expected. A corresponding percentage by the American Men Select Table was 25.91. There were death claims on eight policies for a total of \$31,500 of insurance, six of the policies being less than one year old at time of death. The percentage of deaths for the first policy year was higher than for subsequent years but still very satisfactory."

There have been eight deaths, as follows: cancer, 1; typhoid, 1; suicide, 2; railroad accident, 1; heart disease, 2; hypertrophy with hypostatic congestion of the lungs, 1.

Of the cases dying of heart disease, one gave a markedly abnormal reaction and was accepted only because he was a member of a considerable group of standard risks. The second was a man of 48 with irregular rhythm due apparently to extra-systole. His reaction was normal. He died about 4 months after examination of apparent sudden circulatory collapse.

The case dying of hypertrophy and hypostatic congestion (death report diagnosis), was examined in June, 1922, in the early days of the development of the test. Curiously enough, his reaction was hyperactive, of a type which would now lead to declination. Naturally, our standards of interpretation have varied since then. He died 4 years and 3 months after examination.

The other cases gave normal reactions.

We have heard of three deaths among the declined cases. One, a man of 56 with hypertension (160/94), gave a hypotensive, hypertensive reaction, and died 3 months later of

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cerebral thrombosis. Another, a man of 40 years, had signs of organic heart disease and gave a hypoactive reaction. He died 3 months later of sudden circulatory collapse while hunting. Neither of these two cases would have been acceptable for standard insurance, if the test had not been given. The third case, a man of 54 years, with blood pressure of 142/86, about three extra systoles per minute, and a normal chemical and microscopical urinalysis, gave a hypoactive reaction. He died of endocarditis, secondary anaemia and nephritis, about 7 months later. Without the test, he would have been considered for insurance.

Dr. Hobbs—Dr. Wells.

Dr. Wells—Every thought that I have had on this subject of Dr. Grosvenor has been so admirably covered by either Dr. Cook or Dr. Frost in their discussion that I ask to be excused from any further discussion.

Dr. Hobbs—Is there any other individual opinion of the discussion?

Dr. Toulmin—I would like Dr. Dwight's opinion as to mortality ratios.

Dr. Dwight—25% of the American Men's Select is a little over our own company's, one-half of our own company's first five years' experience during the same period.

Dr. Hobbs—Any other remarks?

Dr. Huston—I just want to mention that the Association of Roentgenologists have a pamphlet giving the names of all of their members who are well qualified to do the work mentioned by Dr. Cook and this can be obtained by the Medical Directors of the different companies. We have such a pamphlet and have obtained the services of very competent men in this work.

Dr. Hobbs—Where do you get the list?

Dr. Huston—Through the association. You can get it either from the Secretary of the society or through their St. Paul office.

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Dr. Hobbs—I think it would be a good plan to have this information if any of our companies wish to avail themselves of the services of a reliable Roentgenologist.

In looking over Dr. Grosvenor's paper, the results impress me, as far as I can remember, as being closely in accord with our own, except, perhaps, in those cases which give a history of acute rheumatism and those cases of hypertrophy. I think our experience is not as favorable, as I recall it, as the experience of Dr. Grosvenor. In the main, the figures correspond to a remarkable degree.

Dr. Grosvenor, will you kindly say a few words in ending the discussion?

Dr. Grosvenor—Our methods may be crude, but we confirm them with mortality facts. The mortality of your company, the New York Life, are closely allied to our own. Different standards of selection enter into that.

As to the value of any test which may be found efficient in determining the functioning power of the heart; to me such tests are of value not alone as a matter of the consideration of murmurs, but as to the general physical standard of the individual in a great many cases. I think, though, we shall have to cling to our own viewpoints based upon statistical evidence for some time yet.

Dr. Cook spoke of the possibility of accepting at standard rates some of the cases at the younger ages. When I first studied these figures a year or so ago, I was somewhat surprised to find that the mortality—I think I commented on it here—was heavier at the younger ages of entry and quite markedly so and at the older ages of entry. In discussions with Dr. Hobbs and Dr. Russell, I learned the same trend was apparent in some of their studies and my company has given some leeway in the rating of an applicant under 30 and we have ceased that practice.

I don't know that there is anything more that I can add.

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I am very much obliged to both Dr. Cook and Dr. Frost for their learned discussions of my experience. I think Dr. Cook's suggestion of the X-ray may be of assistance to us all. A good many of us are using it in an occasional case. Later on we may use it more generally.

Dr. Hobbs—With all that has been said at different times, it seems to me that there is a tendency to try through some means, some test, to separate and cull out those murmurs. We can leave out the words mitral insufficiency or leave out regurgitation, if we know where the murmurs are and how to find them and if they are present through the test, no other impairment being present and the risk being a good risk in every other way and the occupation being all right; and in the younger ages there is a tendency to see if they are not insurable at standard rates. I think there is quite a tendency, as I said, to do this if possible. The question comes up what test to use, and the cardio-respiratory test seems to fill the bill. Other companies may have other tests more simple than this. Whatever the test be, there is that feeling that we can separate into different groups these murmurs found at the apex, systolic, and with very little or no transmission. I believe that this paper will perform an addition to our knowledge on the matter.

We will now proceed to the consideration of the next item on the program, "The Pulse in Life Insurance," an allied topic, and I will ask Dr. Patton and Dr. MacKenzie to let us hear from them in regard to it.

Dr. Patton—I want to preface my remarks by saying that the able assistance of Dr. MacKenzie, who did most of the preparatory work in connection with the paper and edited what we have obtained from the literature, was of great value. We were very sorry to disappoint our President in that we cannot give any statistics from our own records. Our records only go back along these lines for about four years, which is insufficient from an insurance standpoint.



THE PULSE IN LIFE INSURANCE

by

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*Medical Director*

and

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This is not the first time that abnormalities of the pulse have been called to the attention of this Association. Despite that fact we feel no apology is necessary for again bringing them before you, for we are all constantly confronted with the very real question of what is the probable cause of some reported departure from normal pulse rate or rhythm. Your frank discussion of the opinions and ideas expressed is desired, and, should a clearer understanding of the significance of these abnormalities result in a more definite selection, the writers will derive satisfaction from the thought that their efforts were not without some measure of success.

Sir Thomas Lewis in his "Clinical Disorders of the Heart Beat" defines them as follows:

- ✓ 1. Alternation of the heart is a condition in which the left ventricle, while beating rhythmically, expels larger and smaller quantities of blood at alternate contractions.
- ✓ 2. Auricular flutter is a condition in which, as has recently been shown, the contraction wave follows a circular and never-ending path in the auricle, the complete circuits being accurately repeated usually from 260-320 times per minute in different subjects.
- ✓ 3. Auricular fibrillation is a condition in which a wave

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circulates continuously in the auricle, but more rapidly than in the case of flutter, the rate being about 450 per minute; the movement is irregular in that the same path is not followed precisely from cycle to cycle.

4. Extra systoles or premature contractions: Contractions of the heart which disturb the rhythmic sequence by appearing early and in response to pathological impulses.

✓ 5. Heart block: An abnormal heart mechanism, in which there is delay in, or absence of, ventricular response to the auricular impulses. Non synonymous with Adams-Stokes syndrome, as the majority never have fits.

✓ 6. Simple paroxysmal tachycardia: A condition in which from time to time the normal mechanism is interrupted by a series of rapid and regular beats, varying in rate between 100 and 220 per minute, the series starting and ending suddenly.

7. Sinus irregularities: Irregularities of the heart which are produced by interferences with the rhythmic impulses at the seat of their discharge.

We purpose confining our discussion to bradycardia, tachycardia, sinus arrhythmia and premature contractions. These constitute an overwhelming majority of pulse variations demanding our attention. Paroxysmal tachycardia, auricular flutter and auricular fibrillation might in some cases be given favorable consideration, had our examiners the skill and instruments requisite for satisfactory examination and diagnosis. Very rarely are these available and it has been our practice to decline all such cases. Heart block in any of its forms and alternation of the pulse seem to be the result of its conditions precluding insurance. They have also, when reported, been declined. The first four abnormalities mentioned

seem to us to admit of accurate diagnosis and reasonably satisfactory prognosis where sufficient care is exercised.

Before proceeding to a discussion of these, however, we will consider the functional exercise test, for the reason it has seemed to us very helpful in estimating the seriousness of changes in rate and rhythm. Various methods such as climbing stairs, hopping on one foot, stepping off and on a chair, squatting a number of times and bending movements have all been tried in an endeavor to determine the response of the heart and circulation to exertion.

Our preference is for bending movements as they seem more convenient for both applicant and examiner, disturb the applicant's balance less and therefore are less embarrassing for him, are quite as enlightening as any of the others, and we think they are rather more likely to give reasonably accurate results. The feature which appeals to us is that they probably promote flow of blood through the liver and splanchnic vessels with less tendency to flooding of that area, due to a more marked pumping effect than is obtained by the other exercises. As a result the volume flow per minute to the heart is greater. The output from the heart will therefore be greater and the call of the muscular system better met.

When we first began using this functional test we attempted not only to get the pulse rate but also to have the blood pressure taken at the same time. We were very soon impressed with the fact that it was quite beyond the ordinary means of examination to secure satisfactory blood pressure and pulse records in the time required, namely, two minutes. We also gained the impression that the blood pressure was not as accurately recorded as was the pulse rate. Not being able, for obvious reasons, to have two distinct tests—one of the blood pressure and the other of the pulse—we finally concluded to confine ourselves to pulse rate before and after exercise.

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We are not unmindful of the fact that there are very divergent views regarding the value of this test. Vaquez goes so far as to say:

"Recently there has been a great flourish of trumpets over the method of estimating the functional efficiency of the heart by the extent and duration of the acceleration of the pulse produced by exercise. \* \* \* This test, apparently abnormal in subjects free from all cardiac mischief, may on the contrary be normal in others whose heart is on the verge of failure. \* \* \* Moreover, how can one imagine that acceleration of the heart after exercise could be the sole criterion of its efficiency when we know this acceleration is regulated by many extra cardiac and intra cardiac conditions, that in one and the same individual it depends from hour to hour on digestive phenomena and from one day to another on the general health; that it differs from one to another according to the dynamic nervous state of the individual, an idea too often forgotten in physical training? This method, inaccurate in principle and inconstant in results, can give no useful information" (1).

Were we anxious to find support in condemning this test we would probably have difficulty in discovering anything which would more sweepingly consign it to the scrap heap. The following also seems to support the quotation just given:

"Numerous attempts have been made to find a standard for estimating the heart's efficiency. \* \* \* Such tests and the conditions under which they are employed are just as incapable of bringing to light the functional efficiency of the heart as the tests used to find the functional efficiency of the stomach and kidneys" (2).

Such statements, particularly the one last quoted and coming from the late Sir James Mackenzie, demand very serious consideration and we think some temerity is necessary to disagree with them.

We cannot help feeling, however, that in spite of these statements there is merit in the test and we are not altogether

unsupported in our belief. During the late war when some means of determining the functional efficiency of soldiers was necessary, the exercise test was resorted to and extensively used, it would seem with very satisfactory results, by a no less prominent and competent observer than Sir Thomas Lewis.

Some statements by Sir Thomas Lewis are as follows:

"When a healthy man takes exercise, and this exercise is sufficiently stressful or prolonged, he becomes aware at the time of the effort, or after it has ceased, of certain symptoms and he presents certain physical signs" (3).

These signs and symptoms are largely proportional to the individual's general physical condition. They are the signs of fatigue to greater or lesser extent and we think the degree is largely to be attributed to the condition of the circulatory system.

"Symptoms produced in normal subjects by excessive work are produced in the patients in lesser amounts, the smaller the amount of work required the more severe the malady" (4). "The index of ill health is the relatively small amount of work performed which will elicit discomfort" (5). "If there are no clear physical signs of disease, judgment should form itself purely on the observed reaction to exercise" (6). "For upon his (the examiner's) knowledge of the untoward signs of these reactions the efficiency of his work will depend in very large measure. No man who has cardiovascular disease in any degree will pass the simple vascular test. Some few early cases of mitral stenosis and aortic disease will pass all exercise tests satisfactorily and can only be eliminated by physical signs. Such men possess a relatively healthy myocardium" (7).

Speaking of his more strenuous test, which is the bending test we use but with the addition of a weight of twenty pounds, he states:

"A healthy man (aged 20-35) of sedentary habit, and to

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that extent 'out of condition,' can accomplish this lift 30 to 60 times without stopping, but at the end he will be 'blown' and will show clear signs of breathlessness and fatigue. Unhealthy men, 'effort syndrome' cases especially, will show clear signs of distress when far less work has been accomplished" (8).

Speaking of graded physical exercises as a test of fitness he states:

"To pronounce capacity after testing their (returned soldiers) powers of physical endurance by exercise tests of shorter or longer duration is possible in precisely the degree to which these tests approach the crucial test in severity. The plea which is urged is a plea for a wider consideration and trial of exercise tests as the chief means of grading generally, but of grading convalescents in particular, to various types of work or duty" (9).

The late Selian Neuhof, writing of functional efficiency tests and after unfavorably discussing the blood pressure tests, states:

"Far simpler, more readily applicable, and apparently as accurate as the above tests, is the usual clinical method of observing the patient during various forms of exercise, standing, walking, bending, etc., and of estimating the cardiac reserve by noting the rapidity of pulse and respiration, dyspnea and such subjective sensations as discomfort and exhaustion" (10).

Let us say here we do not base our full estimate of the value of the exercise test on pulse rate alone as influenced by exercise, but also take into consideration other symptoms showing impairment of respiration or circulation. Owing to the severe criticism to which this test has been subjected, we wish to give other quotations in support of our attitude.

Cotton, Rapport and Lewis report:

"The pulse rate is invariably highest at the cessation of exercise and declines, at first rapidly, then more gradually, until the normal rate is reached or passed.

"There is the general tendency for the rise of the pulse to be greater with the greater effort, and some series of curves show uniformity in this respect, but it is as usual, if not more usual, to find no material change in the rates until the full effort is approached.

"Within certain limits of effort, it may be said, that the blood pressure rises are controlled more by the actual amount of work done, the pulse rate more by the rate at which work is accomplished.

"It is clear that if we choose a given amount of work as a stimulus, and apply this stimulus to healthy controls and to our patients, the latter react to this stimulus in an exaggerated fashion. The pulse rate rises much higher than in controls, and the high rate is longer sustained, the blood pressure rises higher and the raised pressure is longer sustained than in controls" (11).

Meakins and Gunson state:

"The increase of pulse rate immediately after exercise is relatively more marked than in normal cases, the average maximal rates being 131 to 110.

"In all these cases the relation between the duration of the palpitation and the return of the pulse rate to normal was very striking. The longer the pulse rate took to return to normal the more persistent was the palpitation. In many patients the palpitation ceased suddenly, and in these cases the pulse rate returned to normal with a sudden drop of 20 to 30 beats per minute, and this drop coincided with the cessation of the palpitation.

"On analyzing the severity of the symptoms produced by this simple exercise it was found that those in whom the pulse rate returned to normal within one minute had little or no distress, while those in whom the pulse took longer to return had more marked symptoms. In fact, the severity of the symptoms and the length of time it took the pulse to return to normal were in direct ratio.

"Thus 72 per cent of the cases in which the pulse returned to normal within one minute were eventually able to do all the exercises without symptoms.

"The twenty patients in whom the pulse rate did not return

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to normal within one minute accomplished the exercises indifferently" (12).

Norris also appears to consider exercise of value for he makes this statement:

"Our bodement must also include a careful inquiry into symptomatology. An answer to the question, 'What can he do?'—without fatigue, pain, dyspnea, cough, cyanosis or edema—will shed the light of truth upon many a doubtful case" (13).

Again McLester seems to think it of value as indicated by the following:

"Of greatest prognostic importance, no matter what the cause of the tachycardia, is the degree of discomfort and distress which the patient experiences after exertion. To observe closely the manner in which the patient as a whole reacts to physical effort; to watch for signs of early exhaustion, such as dyspnea on slight exertion; and to discern other signs of myocardial weakness such as slight edema; these are the only means of detecting impending cardiac failure" (14).

Sir James Mackenzie, in discussing heart failure and response to effort, we think in a measure supports our contention, for he states:

"The view was gradually evolved, first, that the symptoms of heart failure are not to be found on examination of the heart; second, that the early symptoms are only to be found when the heart is compelled to respond to effort, and that they are not exhibited by any physical sign, but by the patient's sensations of distress; third, these sensations of distress are of two kinds—those provoked by the heart muscle, which result in pain, and those provoked by a defective output of the heart, which result in symptoms produced by the organs which suffer from the defective output, the chief of these being the symptoms produced by the respiratory system" (15).

If there is any man who in his writings has laid stress on evidence of heart impairment through failure of the organism to respond to exercise previously accomplished with perfect



ease, that man is Mackenzie. If the above is not an acknowledgment that the heart does respond to exercise and that when it fails to respond there is distinct evidence pointing directly to myocardial failure; we are at a loss to properly interpret his statement.

Wiggers, discussing functional disturbances of the heart and circulation, says:

"It is the fundamental function of the heart to ensure at all times a capillary blood flow that is adequate to meet the nutritional and respiratory demands of the body cells. When, for any reason, these demands are increased as a result of augmented metabolism or when the composition of the blood is so altered as to jeopardize these needs, it becomes the function of the heart to increase the minute flow through its adaptive power. The fundamental mechanisms through which this is accomplished are not developed to equal degree in different individuals, however, and, in consequence, the reactions are in one case distinctly physiological in nature and in the other more apt to border upon the pathological. Thus, it happens that in reaction to such influences as exercise, changes in barometric pressure, extremes of external temperature, etc., an occasional heart fails to meet the test and gives rise to the so-called functional disturbances.

"It is quite clear, therefore, that in order to evaluate the pathological physiology of such reactions, it is necessary at all times to compare them with the average run of individuals which are arbitrarily designated as the 'normal'" (16).

Evidently, and this is emphasized by all, there are great differences in the response to exercise manifested by different individuals, a point we have tried not to forget in considering the test. We recognize that Mackenzie's emphasis is placed on a comparison between present and former capacity in the same individual and for that reason a great deal of the error that may creep into the estimation of the exercise test is eliminated. We think, however, that the great majority

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do respond in a somewhat similar way to exercise and that way depends very largely on the state of myocardium.

During the late war the functional test was practically forced on the medical corps through the necessity of determining physical fitness. Life insurance is somewhat in the same position and our experience with thousands of cases in which this test has been used has convinced us that hearts showing other evidence of impairment do not give as satisfactory a response to the test as those in which similar impairments exist but without as yet manifesting any evidence of failing compensation.

Barringer believes the capacity for physical exercise is a valid criterion of the heart's efficiency because the capacity for exercise depends essentially upon the heart's ability to increase its output provided the lungs, nervous system and skeletal muscles are functioning normally. An essential function of the heart is to furnish blood in an adequate amount to a working muscle.

"How can we be certain that a given amount of exercise can be tolerated or performed without overtaxing the heart? There are subjective symptoms of dyspnea, palpitation, fatigue and precordial distress, and objective signs of the dyspnea, facial color and expression and return of the pulse rate to normal in a few minutes. If these symptoms are marked we are uncertain as to the degree of the toleration. A delayed rise in the systolic blood pressure and a prolonged fall indicate lack of toleration.

"It is necessary to state the type, amount and time of exercise used in each case" (17).

Dr. T. Stuart Hart stated at the 1924 meeting of this Association as follows:

"Personally, I feel I derive the greatest amount of information from a simple exercise test, *i. e.*, running, hopping or stair climbing. I do not believe in using a fixed amount of exercise for all individuals whether measured by the number

of steps or foot-pounds, since it is obvious that 100 hops for example will put a very different tax on the trained athlete and the man of sedentary habits. Exercise should be adapted to the individual so that it will be severe enough to produce a slight degree of dyspnea. The appearance of the applicant during this test with a determination of the heart rate before, immediately after exercise and after two minutes' rest will give one as much information as can be gained by employing the more elaborate methods.

"There is another method of estimating the functional capacity of the heart which, in my experience, is of more value than any other, that is the information secured from a carefully taken history. If, in the ordinary activities of life such as stair climbing, running for trains, carrying hand baggage and the like a man develops no symptoms which suggest cardiac insufficiency one may be pretty sure that the heart muscle is efficient" (18).

Brittingham and White state:

"The effects of these functional tests may be measured by changes in the pulse rate and systolic or diastolic blood pressure. \* \* \* Both the extent of the change and the time during which this is sustained being taken into consideration. \* \* \* The results vary in different persons with the type of nervous make-up and general physical condition, as well as with the cardiac power or reserve."

The following are some of their conclusions:

"The results of vital capacity and exercise tolerance tests are in agreement on normal cases but not on pathologic cases.

"These tests are at variance in fourteen per cent of the cases where both were used.

"Judgment in the interpretation of the general reaction of an individual to the habitual activities of his or her life affords the physician more valuable information, as a rule, about the circulation than the use of any functional test" (19).

Dr. Probst used body bending movements twenty times in forty-five seconds in 100 males aged 17 to 53 with 66 mitral regurgitation, eight mitral stenosis, fourteen aortic regurgita-

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tion and twelve combined murmurs, but none with signs of heart failure. Only three per cent reacted to the exercise test by the so-called abnormal blood pressure response. He states:

"The tardy return of the pulse to the pre-exercise level is constant enough in organic heart disease to entitle it to consideration as a test of cardiac efficiency" (20).

We have spent a good deal of time in discussing this test because we are so impressed with the necessity of some means of determining functional capacity and because this seemed to us after experience to be the one most satisfactory.

- The test as used by us consists of thirty bending movements (attempting to touch the floor with the fingers, arms extended, legs extended and body flexed on thighs) in one minute. The number of bending movements to be given, however, is left to the judgment of the examiner. Perhaps we can best explain it by quoting direct from our little pamphlet, "Heart, Pulse, Blood Pressure and Heart Form," which has been sent to our examiners:

"We depend upon the rate and the character of the pulse under varying conditions. Pulse rates which do not return to the original count in two minutes after exercise suggest myocardial impairment.

"The pulse should be carefully counted—applicant seated—for fifteen-second periods until two consecutive periods give the same rate and this rate should be recorded. Exercise is then instituted—applicant standing, knees kept extended—by having him extend his arms above his head and then bring them forward, while flexing the body at the hips in an attempt to touch the floor with the fingers at the rate of one complete up-and-down movement in two seconds. The number of bendings will depend upon the age, sex, occupation, habit as to exercise and general physical condition of the applicant, and must be left to the judgment of the examiner, who should always record the number.

"The exercise must, however, be sufficient to make a real

demand upon the applicant. We have found that the number requested by the examiner is frequently far too few. Thirty bending movements should not produce respiratory or circulatory embarrassment where the heart muscle is competent, especially under age 40. The pulse should be counted immediately following the exercise, applicant seated—and again in two minutes. The rate and the character are to be noted at each counting and recorded. When the rate does not return to the original count in two minutes the time required for such return should be stated."

Two points we want to emphasize. The first is the necessity of gaging the exercise to the age, sex, occupation, habit as to exercise and general physical condition of the applicant; and the second, and more important, is that the exercise must be sufficient to throw a real strain on the heart. The giving of five, ten or fifteen bending movements to a young person of good physique and active physical occupation is to accomplish nothing beyond unnecessarily annoying him. We think if any applicant responds satisfactorily to thirty bending movements taken in sixty seconds, we need not have much fear as to the efficiency of his myocardium.

"The greater the functional efficiency of an organ the more rapidly it will recover its equilibrium after excessive effort" (21).

It is evident that when exercise is instituted all the muscles that are brought into play immediately demand an increased blood supply and register that demand by excitation of the sympathetic system, resulting in acceleration of the heart.

"Acceleration of the heart is a complex phenomenon, depending on at least four factors, excitation of the accelerator nervous mechanism, either intracardiac or extracardiac; diminution of peripheral resistance with consequent quicker expulsion of the blood; incomplete diastole resulting in incomplete filling of the ventricles; and, finally, lack of energy of the systole determined, not by an obstacle, which would have the contrary effect, but by weakening of the energy of the ventricle" (22).

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Not alone do the peripheral muscles call for more blood but as soon as the heart begins to increase its work it also registers a call through its sympathetic fibres, the response being returned in the same manner as from the skeletal muscles. Exercise, therefore, does not directly increase the rate of the heart muscles but indirectly, through its afferent sympathetic nerve and its genetic system. The demand for more blood, however, means a greater output per minute. This is usually met by acceleration, but it may be met not so much by acceleration as by increased filling of the organ with a greater output at each systole. In the latter case reaction to the test will not be so pronounced. The manner in which it adjusts itself, however, will be dependent not alone on filling of the right ventricle but also upon the nerve control of the vessels and heart in whatever way accomplished. It does seem that in the last analysis the brunt of meeting the emergency falls upon the heart muscle, whose efficiency is indicated by variations in the pulse at the time.

Is it not within the bounds of possibility and even reasonable to assume that if fatigue or irritability occurs in the skeletal muscles through exercise, and that when fatigued or irritated they show these through alteration in response, the heart muscle may also react in the same way and manifest irritability or fatigue by abnormal increase in rate or delayed return? There are exceptions to all rules and doubtless are exceptions here, but based on the law of averages we feel very confident in asserting that an impaired myocardium will not respond to exercise as well as will a perfectly normal one. As pulse rate and rhythm are altogether the result of ventricular action, we believe that changes in the pulse may well express in the vast majority of cases what is happening in the ventricle and that when the ventricle is impaired the pulse will, under exercise, respond in a less satisfactory way.

\* It has been our custom to consider a rise of thirty beats a

minute as the maximum increase in rate which should occur under perfectly normal conditions. This idea was somewhat confirmed by an analysis we made several years ago of five hundred cases in which the functional test had been used, and the rate of pulse given before, immediately after and two minutes after the exercise. The pulse also returned to normal within two minutes. Where the pulse has not returned to normal in that length of time we question if we do not condemn the myocardium. A slow return is found in some cases that are apparently perfectly satisfactory in all other respects. In those rare instances we disregard this sign.

Not only do we regard this test as valuable in estimating the ability of the heart to cope with increased strain, but we also find it useful at times in differentiating between rapid pulse due to nervousness and that due to more serious conditions. Again it often happens that the pulse, which is found to be somewhat rapid prior to the exercise, increases very little with this effort but is distinctly slower after the lapse of two minutes. This seems to us to be rather a favorable indication and we think that the rapidity of the pulse was probably due to nervousness, which was overcome when the applicant's attention was focused not so much on himself as on the exercise that was taken. We, therefore, regard such a reaction with favor. It has also seemed useful in pointing to irritability of the heart or to what is described by Lewis as the "effort syndrome."

• A jump of forty to fifty beats per minute immediately after the exercise, followed by a slow return, is, we are confident, a sign that cannot be regarded lightly, and if full information concerning the heart has not been obtained we would not care to act favorably on the case until we had secured all the particulars possible. Where irregularities in rhythm exist it seems to be our only means of determining their seri-



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ousness, and we base our interpretation of the significance of the irregularities on their reaction to this test.

One word before going on towards a discussion of the irregularities. It may be thought from the above that we believe the myocardium is always involved where there is failure of satisfactory response to exercise. We would like to offset the possibility of such an impression remaining by saying that in our judgment a perfectly healthy myocardium—and by healthy we do not necessarily mean strong or vigorous, but one in which no recognizable pathological condition exists—may have such a strain thrown on it through impairment of some other organ or alteration in the blood that it is unable to cope with these changes and meet the added strain of the exercise without showing the effect. We would, therefore, like to urge first, the necessity of considering the exercise test among the methods of evaluating the heart; second, that failure of the organ to properly respond to this test does in all probability mean depreciation somewhere in the economy, most likely in the heart muscle; and third, that good response is strongly suggestive of the ability of the myocardium to take care not only of its daily task but of a considerable degree of increased strain in a satisfactory way.

It will be noted that no reference has been made to the functional test advocated within the past few years by the New England Life Insurance Company (23).

Although this test does put a strain on the myocardium it is not strictly speaking an exercise test in the sense in which we are using that term. While it is highly recommended by them after considerable experience and analytical study we have not as yet used it sufficiently to warrant our comment. Our experience with it up to this time leads us to think it is a far more searching test than is that where changes in the pulse alone are considered.



A little over twenty-four years ago Mackenzie wrote regarding pulse irregularity:

"There are no deviations from the normal character of the pulse so easy to recognize as irregularities in the rhythm and yet there are no abnormalities whose symptoms are so surrounded by mystery, whose description in medical literature is so hopelessly confused and whose diagnostic significance is so completely misunderstood" (24).

Since the above was written vast changes in our knowledge of the anatomy and physiology of the heart have taken place and our understanding of the significance and diagnosis of these changes has clarified, chiefly as a result of the work of the author just quoted, so that today we are in a much better position to understand the causes of these irregularities and to make a more accurate differentiation.

The Medico-Actuarial Investigation in 1914, reporting on the mortality of pulse departures from normal, showed that for Class 29, irregular pulse, the mortality was 95%, and explains it by the fact that:

"This low mortality is probably due to the inclusion of many cases wherein an irregular pulse was found on only one out of two or more examinations" (25).

With such a mortality the significance of irregularities as found on examination seems insignificant. This is especially true for the reason that it seems to have been the practice, during the period when the statistics from which these figures were derived, to write back to the examiner when an irregularity was detected with the request for re-examination. If the second report showed no irregularity the case was accepted. If it was present the case was postponed or permanently declined.

Intermittent pulse, Class 30, shows a slightly higher mortality—113%. There is some increase in mortality over that shown for irregularities. The surprising feature is that there

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was not a greater increase, as we believe these cases were selected in much the same way as were the irregularities. The figures do point to the fact that we are on more dangerous ground when dealing with intermissions. We would have expected a higher mortality and think the favorable percentage shown may have been due to a somewhat more severe selection.

The mortality for pulse rates between 90 and 100, Class 31, Group K, 172%, we believe exaggerates the significance of such a pulse rate, for where found at the time of examination the mortality was 172%, but where there was one record within ten years of application it was 145%. A significant comment is made:

"The death rate from heart disease and pneumonia and especially from tuberculosis of the lungs was distinctly above the standard."

To our minds the increased mortality was not because of increased pulse rate but because of failure of the examiner to detect the reason for the increase. It might, of course, be argued, and probably would be by many, that all such pulse rates demand an increase in rating or a rejection. Undoubtedly some of them do. There is a large percentage, however, that, with our present knowledge, can be shown to be the result only of excitement or emotion at the time of examination, therefore of no serious significance and insurable at standard rates.

What has been said for pulse rates under Class 31 may be applied with greater reservation to Class 32, where the rate ran over 100 and in which the mortality was 205%. Such mortalities as given are altogether too high to be disregarded, and before concluding the increased rate is due to some fleeting condition, very careful investigation of the general state of health is necessary, thorough questioning and examination for evidence of disease, particularly tuberculosis,

as it is stated the "death rate from tuberculosis of the lungs was high but did not account for the entire excess mortality."

Since 1914, when the investigation just mentioned was completed, we have learned a great deal about focal infections and have also come to understand more fully the effect on the myocardium of acute infections, particularly rheumatism, chorea, pneumonia, influenza, scarlet fever and diphtheria. We are consequently in a better position today not only from the viewpoint of increased knowledge of the heart and its control, but also of the causes of its impairments, to make a more equable selection than was possible when the data was secured upon which the Medico-Actuarial report was based. In the light of present knowledge, we believe it is now possible to accept irregularities in rate and rhythm and obtain lower mortalities. The figures above quoted therefore should, while a warning, not be regarded as a true index to the seriousness of pulse abnormalities but rather as exaggerating their significance, provided we adhere to the selection we are now able to make.

The changes in our knowledge of the anatomical arrangement of the heart and of the innervation and control of that organ have been so great in recent years, and the present day interpretation of changes in pulse rate and rhythm are so dependent upon this knowledge, that we want to very briefly refer to them as what will be said is based entirely upon this altered viewpoint.

When, twenty-four years ago, Mackenzie wrote about the "hopelessly confused" state of knowledge, some of the material upon which the actuarial report above referred to is based was being accumulated. It was the belief at that time that changes in rate and rhythm were due to direct action of the cardiac nerves, upon the heart muscle or to disease of the muscle itself. In recent years it has been learned that the heart is really composed of an organ within an organ,

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the contained organ being described by Mackenzie as the genetic system, which has its origin in what is known as the sino-auricular node which lies just outside the heart at the junction of the superior vena cava and right auricle. Connected with it by neuro-muscular bands and beginning in the region of the coronary sinus is the auricular ventricular or A-V node, which gives rise to fibers that form a bundle; a "distinctive system of muscle fibers enclosed within its own sheath." This lies beneath the endocardium and is known as the His Tawara system. It divides into two branches. The right passes to and ends in the musculature of the right ventricle; the other, the left, terminates in that of the left ventricle.

This is the controlling mechanism of the heart which is capable within itself of generating, regulating and conducting impulses, but normally it is influenced by the vagus and sympathetic nerves, the sympathetic and right vagus nerves being distributed to the S-A node, the left vagus influencing to a greater degree the A-V node. It is through this system only, however, that the cardiac nerves affect the heart muscle. They, therefore, give rise to variations in rate and rhythm not by direct action on the muscle but through the genetic system.

The point we wish to emphasize here is, and on this point is based recent understanding and interpretation of changes in the pulse, that all changes in rate and rhythm of the heart come to it directly, and only, through the genetic system. When this is understood, changes in rate and rhythm are more easily explained. On this point all modern cardiologists seem to be agreed. When, therefore, some departure from the normal in pulse rate or rhythm occurs, we no longer think of the heart muscle as the cause but immediately look to the genetic system for an explanation.

Vaquez says:

"Every motor excitation must travel the auriculoventricular path of the bundle of His, and if, from any cause, this passage is made difficult, there result disorders of rhythm which, for this reason, are called disorders of conduction" (26).

To again quote Mackenzie:

"It will be found that all irregularities are due to an increase or decrease of activity of the different parts of the genetic system, or an interference with the function of control (27).

"From these experiments it is seen that the S-A node is quite independent of and indifferent to the work of the auricles and ventricles, and that their distension has no direct effect upon the genetic system so far as the rate of the heart-beat is concerned. The fact, however, that the heart varied in the strength of the beat shows that there is within the heart itself a regulating influence, and this regulating influence can only be due to the genetic system" (28).

The sino-auricular node is "the pacemaker" and entirely controls under normal conditions the rate of the pulse and minor changes in rhythm. It will be seen, therefore, that change in rate—true bradycardia, true tachycardia and sinus arrhythmia are produced entirely through this node and are not in themselves in any way indicative of cardiac defect.

The node is under the influence of the vagus nerve, particularly the right branch. As a result of this influence, the rapidity of impulse formation is decreased when the nerve becomes more active. It is also under the influence of the accelerator nerve (sympathetic) which is the great antagonist of the vagus and which when called into action offsets the influence of the vagus, thereby permitting increased impulse formation. Impulses generated outside the S-A node are invariably abnormal, and it is these ectopic impulses which give rise to the various types of premature contractions and other but still more serious irregularities. The

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character of these irregularities is, therefore, dependent on the location, extent and severity of involvement of the system.

The viewpoint just taken is very different from that formerly held, but is based on present day interpretation of the findings obtained through the use of polygraph, sphygmograph and electrocardiograph. It is almost impossible in our work to avail ourselves of these means of diagnosis. We are, therefore, compelled to differentiate without their use. Such differentiation is nearly always possible in the types of abnormality which we are apt to meet. Apart from purely physical findings, subjective symptoms suggestive of cardiac impairment are vitally important to a proper interpretation of the defect, and second to these is, we believe, a history of previous disease, particularly the acute and focal infections, with a history of such infections, past or present, and with evidence of subjective symptoms, the probable seriousness of pulse abnormalities is materially increased.

We have at last come to the specific abnormalities mentioned at the beginning of this paper.

Bradycardia may be looked on as being present when the pulse rate is below 60 per minute, although the pulse may be much slower. Mackenzie reports having

"seen two cases with a pulse rate of this kind, in one 36 per minute and in the other sometimes lower than 30 per minute. The latter was an acrobat forty years of age and had himself been aware of the slow heart rate for twenty years" (29).

These are interesting exceptions which simply serve to emphasize the fact that a very slow pulse may exist without other evidence of organic change. A pulse under 65 is usually associated with vasomotor instability and is particularly common in hypothyroidism. It may, however, be purely emotional through vagal stimulation or may result from pressure on that nerve.

One of the writers has examined a physician who was in perfect physical condition but who was able through posture to so stimulate the vagus that his heart actually stopped beating for what seemed an alarming period. He could do this at any time at will.

A slow rate may also, of course, be due to one of the forms of heart block. When met with we, therefore, have to satisfy ourselves that no pathological cause exists. This is possible with the exercise of care. In the first place, not only should the pulse be carefully counted but it should be as carefully compared with pulsations at the apex, determined by stethoscopic examinations. Where these are synchronous and regular, we have every reason to feel that the genetic system is intact and the slow rate the result of decreased impulse formation in the S-A node. Again where there is organic change at some point in the main conducting system below the S-A node the pulse rate will not materially increase under exercise, for the reason that transmission of impulses is blocked. Where, therefore, exercise produces an increase in the pulse rate, we are further assured that the conducting system is unimpaired.

There are then three points in the differential diagnosis which we wish to emphasize and which are available to any physician, in his office or at the bedside, namely, increase in rate under exercise, perfect synchronism between the apex beat and radial pulse and regularity of rhythm.

Our limit of approval at standard rates for a slow pulse has been fifty, but we are confident that rates lower than this are quite insurable. We would, however, feel justified in imposing a rating which seemed to us commensurate with the lower rate and other features of the examiner's report.

Tachycardia, the opposite of bradycardia, is like it due to altered impulse formation in the S-A node. It is, therefore, merely an increase in rate, in its mildest form, and is largely a

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nervous phenomenon. The nervous element we think is particularly prone to exist at the time of examination for life insurance. The Medico-Actuarial statistics above quoted, however, seem to point very definitely to the fact that it may at times be due to serious impairment in regions remote from the heart or in the heart muscle itself. This is particularly true in cases of consumption, even in the earliest stages of incipency, and also in hyperthyroidism. General nervous depression or lack of tone with irritability of the accelerator nerve may also well be factors in its production. Its presence therefore always suggests not only a retaking of the pulse but searching inquiry and re-examination to determine the possible presence of some abnormal condition giving rise to it.

It may be differentiated from the abnormalities with which it is most likely to be confused, especially paroxysmal tachycardia, by the fact that the rate increases and decreases gradually, whereas paroxysmal tachycardia begins and ends abruptly. Simple tachycardia is also influenced by exercise. Paroxysmal tachycardia and auricular flutter are not so influenced and the irregularity accompanying auricular fibrillation is increased, while the rhythm in the condition we are considering is not altered. True tachycardia is also influenced by posture, the rate decreasing when the applicant lies down. This is a distinctive sign. We have often found that under the exercise test, as has been before stated, the rapid pulse decreases as the result of transferring concentration from self to the exercise. Variability in the rate is also suggestive, as no variability is found in paroxysmal tachycardia or flutter. Furthermore, the rate of the pulse is a very good indication as to the character of the increase, for in true tachycardia it is practically always under 140 and usually not over 130. A pulse rate of 140 always raises the question as to the presence of some more serious condition.

A case recently came before us that we think is of interest



from the viewpoint of increased rate and variability. He had been previously declined by this company on account of a pulse rate of over 100; we believe that a similar condition had been observed several times previously, and that once there was evidence of irregularity, the form of irregularity not stated. These examinations occurred four to five years ago. The applicant is married, 31 years of age and a book-keeper; personal history as to illness negative; physical examination at the time showed sight and hearing good, height and weight 5' 7" and 165 pounds, pulse 118 neither intermittent nor irregular, temperature 98, blood pressure 125 systolic, 87 diastolic, urine normal. The doctor stated:

"I have examined the applicant's heart three different times. He gives no history of any circulatory disturbance; the examination of the heart before exercise on first examination pulse rate 92, after exercise pulse rate 124. Examined the same two months later—pulse rate 118, which had remained for over ten minutes. Second and third examinations—pulse rate was about the same after and before exercise except that I noticed on the third examination, which was twelve days after the first, the pulse rate before exercise was 88 and was irregular. After exercise the pulse rate was 102 and not irregular."

The one and only unfavorable feature concerning this applicant was a somewhat increased but variable pulse rate. The response to exercise in one of the tests suggested no great irritability, the rise being 32. In 1922 our examiner reported the pulse rate 130, after exercise 144, but returning to normal in two minutes, physical condition good. On August 10, 1926, we reconsidered our previous action and approved the case, but found it necessary in view of the present attitude toward these cases to impose a rating.

We are of the opinion that the findings in this case were solely the result of changes in the S-A node stimulation and therefore, purely functional, at least as far as the heart was

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concerned. The examination of the heart four years ago corresponded with present examination, both examiners considering him a good risk apart from the rapid pulse. We believe, at least from a clinical viewpoint, that his pulse rate had no serious significance and that a consideration of all the features in the case justified the conclusion that we were dealing with a sound heart in a sound body. A rating was imposed, not because of any known serious physical condition but because of the history on three or four different occasions of the presence of the same abnormality and the desire to protect the company against the barely possible presence of some defect which as yet has not manifested itself.

Differentiation of increased rate may then be made by the gradual onset and decline in rate, by the fact that the rate does not exceed 140 and is rarely over 130 per minute, by its becoming slower with change of posture from the standing to the recumbent position and by its often disappearing under the exercise test. We, however, wish to again emphasize the possibility of the presence of hyperthyroidism, pulmonary tuberculosis or some asthenic condition which would have been revealed had a more painstaking search been instituted. It is, of course, understood that what we have said above is based on the supposition that the heart itself shows no evidence of abnormality other than changed rate.

Sinus arrhythmia may be disposed of very quickly for the reason that it is easily detected and not of serious significance. It is due to variations in the length of the diastolic period. The systolic periods, however, always remain of the same length.

"There is never an incomplete ventricular systole" (30).

It has its origin in the S-A node from vagus stimulation, may be increased by taking a few deep breaths and will always disappear under exercise sufficient to raise the pulse rate to 100 or over.

"As a rule, if the breathing is held, either in inspiration or in expiration, the variation in rate disappears" (31).

Its presence is never the result of myocardial involvement or of pathologic changes in the genetic system. Occasionally stimulation of the vagus may be sufficiently intense to give rise to prolonged diastolic periods which on superficial observation through apparent intermissions of the pulse may be mistaken for heart block. When the lengthened periods are frequently repeated, marked slowing of the pulse will be present.

"There are rare cases in which a period of standstill is so long that a patient loses consciousness" (32).

We think it can be readily understood how this change in rhythm might, under the emotion or excitement attendant upon life insurance examination, be responsible occasionally for a feeling of giddiness, dizziness, or even slight faintness, which would readily be overcome by exercise.

Mackenzie says:

"Sometimes we come across individuals in perfect health in whom there are long pauses in the pulse, evidently due to the temporary suspension of the activity of the S-A node. I have watched several individuals for ten or fifteen years who exhibited this peculiarity at frequent intervals, and they never showed any cardiac impairment" (33).

When we receive reports of dizziness or faintness during examination the possibility of these being due to the condition just mentioned should not be forgotten.

This arrhythmia was described by Mackenzie as:

"The youthful type of irregularity which is almost always present in children becoming more rare after puberty but may be found in the third and fourth decades or even later."

We feel justified in saying that this type, which is one of irregularity in rhythm but not in force, and which disappears

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following exercise, usually to reappear after the effect of the exercise is past, may be disregarded.

✓ Premature contractions are always ectopic in origin and require more serious consideration than do the arrhythmias already discussed. They were first recorded by Mackenzie in 1892 (34), since which time they have been extensively investigated so that today we have some, though as yet imperfect, knowledge of their cause, production and effect. They are of very frequent occurrence, some authors stating that they constitute about 50 per cent, and combined with sinus arrhythmia 90 per cent of all irregularities.

To again quote Mackenzie, he stated:

"They were found to be rare in the young and a little more frequent about middle life. Over fifty years of age many people exhibit them, while probably everyone over sixty exhibits them at one time or another, or continually" (35).

There seem to be four types according to the location in which the impulse arises, namely, the sinus, auricular, nodal and ventricular.

In the sinus type the whole heart is involved due to an irregular impulse production in the S-A node.

Vaquez states:

"The sinus is not subject to the same laws as the other parts of the heart, notably in that which concerns the refractory period. Ever excitable, it is always ready to respond to an excitation by a contraction and the arrival of a premature excitation merely releases a premature contraction, which is then transmitted to the auricle and to the ventricle" (36).

Auricular extra systoles are of great interest for:

"They are often the prelude to such arrhythmias as paroxysmal tachycardia and complete arrhythmia, which indicate serious disturbance of the function of the auricle.

"Auricular extra systoles too cause certain changes in the rhythm of the pulse, such as pulsus bi-geminus or couple beats, pulsus trigeminus, pseudo alteration, etc. There is

one, however, which they never produce—bradysphygmia” (37).

Nodal extra systoles originate in the junctional tissue and with the two just mentioned require graphic methods for their detection.

The type that particularly interests us and the one most common is the ventricular. It is also the one admitting of diagnosis by simple clinical methods. Premature ventricular contractions may be occasional or frequent and may occur at regular or irregular intervals, in groups or singly. They may be extracardiac or intracardiac in origin, but are always due to impulses, transmitted through the genetic system, which occur prematurely in some portion of the diastolic period other than that which is absolutely refractory.

Mackenzie states in referring to their origin:

“When we consider the various forms of extra systole, we can recognize that as there is an effective contraction of the muscle the impulses must be conveyed to the muscle by a special structure. The extra systole is in this sense an effective contraction even although in some instances the output from the ventricle is small. \* \* \* So far there has been adduced no evidence demonstrating that muscle fibres can originate a stimulus that would produce an extra systole. When muscle fibres contract independently of the genetic system the contraction takes the form of fibrillation. \* \* \* We have no evidence that a muscle can become refractory. \* \* \* The refractory state is manifestly due to the genetic system” (38).

Evidently the muscle in itself cannot give rise to premature contractions but when irritated may stimulate the genetic system to produce them.

The causes responsible for them may be broadly divided into two—first, extracardiac due chiefly to reflex stimulation from the pregnant uterus, stomach or other abdominal organ and second, involvement of the heart itself.

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Vaquez says:

"All diseases of the heart may be accompanied by extra systoles. Sometimes, with uncomplicated valvular lesions, for instance, it is simply a coincidence; or the extra systoles may be caused by failure of the heart. They are, therefore, very frequent in the course of acute myocarditis and in the advanced period of chronic myocarditis and in genuine hypertension. They are then a sign of danger" (39).

In Osler's Principles and Practice of Medicine we find the following:

"Extra systoles occur at all ages and under the most varied conditions but are most common in persons over fifty. There are several classes of cases, the irregularity may be a life-long condition, without any recognizable disease and without any impairment of the function of the heart. This may be a peculiarity of the heart muscle of the individual, who has extra systole for the same reason—physiological but not well understood—as the dog and horse, in which animals it is common. \* \* \* In a second group toxic agents, as tobacco, tea, coffee, or the toxins of the infectious diseases are responsible. Digitalis may be a cause. Even reflexly, as in flatulent dyspepsia, extra systoles may arise. Thirdly, a high blood pressure can set up extra systoles; also change in posture. And, lastly, organic disease of the heart itself, especially myocardial" (40).

To again quote Vaquez:

"There is a disorder which seems of itself to originate extra systoles which may recur almost incessantly with veritable cardiac delirium; this is chorea" (41).

Chorea and articular rheumatism are so closely associated and rheumatism has such a selective action on the heart that it seems to us quite possible, even probable, that premature contractions may accompany or follow this infection.

Evidently then myocardial disease, strain and intoxications are the great causes for this abnormal rhythm, although irritation of organs far removed from the heart may, through

reflex action, be the causative factor.

The diagnosis is usually not difficult. There are intermissions of the pulse occurring sometimes regularly and sometimes irregularly and these intermissions are accompanied by alterations of the sounds at the apex where will be heard, instead of the regularly recurring first and second sounds, four or three sounds followed by a prolonged pause. These four or three sounds together with the pause constitute a period slightly less than two cardiac cycles. Four sounds are heard where the extra systole is sufficiently strong to open the aortic valves but only three with a weaker contraction.

May we digress here to again emphasize, as we tried to do in a former part of this paper, the necessity of carefully comparing abnormalities of the pulse at the wrist with the apex beat? Failure to do this will very possibly lead to serious error.

The pulsations between the extra systoles are regular in rate, in rhythm and force. Where the premature contractions are what is called functional in origin, or due to slight organic impairment, they will disappear on exercise to reappear as the heart again becomes slower.

It seems to us there has been a tendency to regard this irregularity as of minor or no importance. Probably the most outspoken in this respect was Sir James Mackenzie, who said in speaking of the different ages at which they occur:

"We recognize in this view an occurrence similar to what happens with people with gray hairs, and so far as I have seen, I would attach no more gravity to the one sign than to the other" (42).

He followed this with the statement:

"If cardiac impairment is found associated with extra systoles, then the prognosis depends upon the condition of the heart which leads to the impairment, and not upon the extra systole, which at the worst, indicates but a coincident change" (43).

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The opinions of the late Sir James Mackenzie, and others of note, are entitled to every respect, yet, it is easy to over or underestimate the seriousness of any impairment, no matter how careful the observer, when opinion as to the gravity of a condition is based on the number remaining under observation after some years.

We are afraid were we to regard extra systoles in this light we would be courting trouble. It may be all very well for men with special diagnostic ability to so regard this abnormal rhythm. It is quite another thing where we have to trust to examiners whose skill in estimating cardiac conditions is, in the great majority of instances, very deficient. We think anyone who reviews many reports from examiners will be forced to this conclusion. What Mackenzie has said must, therefore, be accepted only in proportion to the skill, or lack of it, with which hearts are examined and assessed.

Hirschfelder has stated that an

"occasional extra systole in an otherwise healthy person whose blood pressure is normal and who does not become especially short of breath upon exercise may be entirely disregarded" (44).

And T. Stuart Hart makes the following comment:

"My personal belief is that there is an underlying slight myocardial defect although in the majority of instances, no such lesion has been demonstrated by the pathologist. In most cases, this muscle damage is so slight that it should not be regarded as an impairment. When these abnormal beats are infrequent or transitory and all arise from the same point in the myocardium (which is usually the case) the applicant should be accepted as a standard risk" (45).

We think the quotations given support our contention that extra systoles are real danger signals, although the majority may not arise from serious impairment. Those due to irritation of some region other than the heart have to be con-



sidered. Before concluding, however, that they do not originate in the heart, very careful inquiry for evidence of disease of one or more remote organs, particularly the stomach, should be made. When they do not have their origin outside the heart the question immediately arises as to the presence of existing cardiac impairment. If they are present following one of the acute infections, we would fear actual myocardial change, probably slight in degree but with future possibilities. Certainly we are not inclined to consider lightly any extra systole which we have reason to think is the result of a cardiac impairment no matter how slight that impairment may apparently be at the time of examination.

We were struck with a statement made by Mackenzie, regarding the occurrence of these premature beats in children. He said:

"In a very extensive examination of children's pulses I have only found two or three instances where the pulse irregularity was of the adult type. When the irregularity did occur it was only for one beat at long intervals. In none of these exceptions could the individual be considered healthy. In advanced heart disease the pulse of children is often irregular and it then assumes the same character as it does in adults" (46).

If the heart that is young manifests this irregularity only when diseased conditions are present, how can we escape the thought that when we meet the same irregularity in more advanced life there is probably some impairment present which as yet is either not detectable or undetected.

To again quote from Osler's Principles and Practice of Medicine:

"The significance of premature contractions is not always easy to determine. They are often temporary, especially in young persons, but should not be regarded lightly. It is wiser to regard them as meaning some pathological change until the contrary is proved than to make light of them and

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recognize the error later. In those who have reached fifty years of age they may be the warning of serious myocardial damage. The patient seen today with extra systoles may return with auricular fibrillation in two or three years" (47).

Vaquez says, in referring to extra systoles:

"Sometimes the irregularities appear only at long intervals on the occasion of some exertion or emotion; or, infrequent in the beginning, they soon recur several times a day for several days in paroxysms separated by quiet periods. In some instances they are truly exasperating in their tenacity and their incessant recurrence" (48).

Further on he states:

"However, with young subjects extra systoles are seldom serious. Often they disappear as they came leaving no trace, although they may recur some years later. Even in this event, contrary to the opinion of Wenckebach, we do not believe that of themselves they are capable of precipitating heart failure. In patients who have passed their fiftieth year, they may have no greater importance, but, as Huchard said justly, any arrhythmia that appears in middle age should be investigated. Often it is the first indication of latent arterial hypertension, announcing the eventual occurrence, though perhaps at a distant date, of failure of the left ventricle" (49).

Craig states:

"The outlook depends not upon the premature beats but upon the cardiac abnormality with which they are associated. On the other hand, they frequently occur in hearts where their presence is the only evidence of disturbance. Here they suggest a nutritional disturbance either temporary or more or less permanent. But they themselves alone are not evidence of serious heart involvement" (50).

We think we have shown from the foregoing quotations that extra systoles do suggest grave possibilities, either present or remote; and that they are usually less serious in the young, probably because of the greater proportion of healthy heart muscle and greater reserve power. As age advances

their gravity increases and over the age of fifty we would want to be very certain as to the general and circulatory conditions before being willing to consider them insurable at standard rates. We also think that their gravity is in some measures suggested by their frequency. There is little doubt that the frequency with which they occur is an expression of the degree of irritation which gives rise to them.

Before concluding that extra systoles are entirely the result of the use of tobacco other possibilities should be carefully excluded. Whether the long continued use of tobacco, by those so susceptible to it that enough irritation is produced to give rise to these extra systoles, may be disregarded is a question we are not prepared to answer. We are inclined to think that the irritation thus produced may, like other irritations, in the course of time occasion structural changes. At least a muscle so irritated would be more vulnerable to disease. An occasional extra systole resulting from tobacco and which disappears following exercise may be disregarded. Where frequently we are disposed to make careful inquiry as to the amount of tobacco consumed, we would want to exclude as far as possible all other causes, and would be somewhat influenced in our estimate by the family history.

Where they are infrequent in number, occur under the age fifty in applicants otherwise normal, and disappear following exercise, we have been in the habit of taking them at standard rates and still feel we are justified in that practice. We try, however, never to lose sight of the fact that when all is said and done they are an expression of the existence of some abnormal condition, temporary or permanent, and if permanent liable to increase in severity as age advances.

We will not take them where there is other evidence of cardiac disease. Undoubtedly there are some cases that could be so taken if the means at our disposal were such that we could obtain an exhaustive examination and thoroughly competent

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opinion as to their origin and probable significance. We are not in so fortunate a position but knowing that they so frequently occur from actual impairment, we cannot under existing conditions feel warranted in accepting them in the presence of heart murmurs, hypertrophy or possible sclerotic changes of the heart or vessels.

It has been a matter of great regret to us that in the preparation of this paper we have been unable to present views based on statistical study of material in our office. This has been impossible for various reasons, the most important of which is that the data on hand is insufficient in amount and does not cover a long enough period to justify drawing conclusions therefrom. At a later date we will have such data and hope it then may be our privilege to present it to this Association. In the absence of sufficient data we have been forced to depend largely on the literature of the subject, as a result of which numerous quotations have been given. We are not aware of the existence of any statistics on abnormalities of the pulse other than those in the Medico-Actuarial Investigation.

We feel sure that there is here a large problem with which we have been, and still are confronted, namely, how to get from the examiners the data necessary for a reasonably safe interpretation of any abnormality which may be present. Pamphlets are read by some, and individual letters on specific cases probably read with care by the recipients. The personal interview is, however, best. This, of course, is not feasible with every examiner but it is possible in the larger centers from which the great bulk of our work comes and in which we think we are most apt to meet with the more serious impairments, so that the personal touch is our principal hope. By personal letters on individual cases we can sooner or later reach all with specific instances. Last, but probably the least effective, though undoubtedly of value, is the issuing

of pamphlets on various subjects, the most important of which is the circulatory system.

#### SUMMARY

The functional exercise test is believed to be of real value in the great majority of cases for determining the functional efficiency of the heart. Age, occupation, habits as to exercise, etc., must be considered and the amount given must be sufficient to throw an actual strain on the heart.

Paroxysmal tachycardia, auricular flutter, auricular fibrillation, alternation of the pulse and heart block are deemed not insurable, at least, under present opportunities for estimating their value.

True bradycardia, true tachycardia and sinus arrhythmia are believed insurable at standard rates.

Premature contractions are always abnormal. Under age fifty they may be insurable at standard rates. Over age fifty they always give rise to grave doubt as to the myocardium being normal. In neither the young nor the aged should they be regarded as lightly as has sometimes been suggested.

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Dr. Hobbs—Dr. MacKenzie, have you anything to say on this subject?

Dr. MacKenzie—Not at this time.

Dr. Hobbs—We will pass on to the discussion of this paper and ask Dr. Martin to discuss the paper first as I believe he has an appointment and it will be necessary for him to leave a little earlier. I am going to ask Dr. Martin to give us his views.

Dr. Martin—I thank you very much, but I would like to hear Dr. Dwight first.

Dr. Dwight—Mr. President and Gentlemen: The consideration of "Pulse in Life Insurance" is of much importance to us all, and questions in connection with it arise in almost every application which comes to our desks. When I agreed to discuss this paper of Drs. Patton and MacKenzie it seemed to me that it ought to be comparatively easy to find something to say, but after reading the very carefully written and admirably expressed matter which was put upon my desk, I find it a little difficult to know just where to begin.

These gentlemen have made no attempt to cover the whole subject and I think that they were very wise in reaching that decision. That portion which they have touched upon, however, they have handled so thoroughly and in such a judicial way as to make it very difficult to "discuss" in the ordinary way. They have given both sides of the question, particularly in so far as it applies to the literature in regard to the test; they have expressed their opinions distinctly and clearly and they have not tried to reach any definite conclusions or to furnish statistics upon which such conclusions could be based.

For these reasons it has seemed that I could serve best by adding to their facts, certain others which were not available to them and so I shall limit my discussion to our addi-



tude towards these tests and in a very limited way give some of the results which we have obtained.

A study of the material of our company based upon all of our business prior to 1905 showed that on the whole these doubtful pulses had been accepted in a generally satisfactory way. Cases of bradycardia, tachycardia, irregular and intermittent pulses were looked upon with considerable doubt. They were carefully selected so far as the knowledge of our Medical Department would admit and the applicant with any of those conditions must have been about right in every other way if he was accepted.

During this period our average mortality had been 74% of the American Table and our results written on individuals accepted with varying conditions of the pulse were as follows:

Pulse under 50	95.2%
" 50-60	72.7
" 60-80	78.6
" 80-90	100.4
" 90-100	128.0
Irregular pulse	72.2
Intermittent pulse	99.4

The best mortality was with a pulse rate of 50-60, gradually increasing in rates up to 100. Above this very few cases were taken—not sufficient to be of any importance. It was noticed that whatever increase in rate occurred above 60 beats per minute were associated with some increase in mortality in late life. When more rapid, as between 90-100, there was an increase in early life as well as after age 50 and in intermittent pulses it was found that the mortality was quite high after age 60.

In those cases which gave a history of irregular, intermittent, rapid or slow pulse but which on examination came within normal limits, the general results were very satisfac-

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tory although the mortality again was somewhat higher than normal after age 55.

Since that time we have made no particular study of our experience, but we have been careful in the selection of individuals with a pulse over 90 and if quite rapid, or intermittent in character, they were offered policies terminating at or about 60 years of age.

This method of handling them is not, of course, satisfactory and certainly is not in accord with our present methods of selection.

We, like the authors of the paper, have been seriously considering the methods by which the efficiency of the heart and circulation might be measured. For various reasons we were not satisfied with the methods employed by the army and navy during the war. They were undoubtedly more or less effective for the purposes for which they were developed, but they were cumbersome and from our point of view unsatisfactory when applied to applicants for insurance of all ages, both sexes and in all kinds of physical condition.

As the authors have pointed out, the reaction to any physical effort depends on so many factors as to be difficult to interpret particularly from evidence furnished on paper. The ability to withstand exercise varies so much even among normal individuals as to make it difficult to regulate and up to the time of Frost's development of the cardio-respiratory test and our application of it to various forms of circulatory impairments, we had discovered no method that we thought it wise to inaugurate as a definite system.

I do not propose to discuss this test at this time, but it has three advantages which to our minds are of great importance.

First. The load which the individual carries is one chosen largely by himself.

Second. It is a measured load which we can compare with the measured results of carrying it, and

Third. We have the opportunity to test not only the pulse before and after, but also the systolic and diastolic pressures before, after and during the test.

In making a study of the results of this test on the pulse preparatory to this discussion, it became apparent that the amount of exercise involved was not as a rule sufficient to seriously influence the pulse rate. As a matter of fact in 1228 cases it developed that in 29+% the pulse was more rapid before the test; in 56+% after the test and in 14+% the rate was not modified. It was also interesting to notice that there was no material difference in this record between the group of cases which we approved for insurance after the test and those which were declined as a result of it.

The influence of the test on systolic pressure was also interesting as it showed that of 1236 cases in 50+% the systolic pressure was higher before the test; in 34+% after the test and 14+% there was no change and again these percentages were not materially different whether the applicant was approved or declined.

Of 1215 cases in which the diastolic pressure was noted we find that 29+% were higher before the test; 50+% after and that in 20% it was not influenced. It is also a fact that these percentages of diastolic pressure were almost exactly the same in the group which was declined and that which was accepted.

Amiral has recently reported that in the cardio-respiratory test the diastolic pressure does not follow the systolic, but tends in the opposite direction and these results furnish some confirmatory evidence along that line.

During the past five years we have been applying the cardio-respiratory test as described by Frost to a large and heterogeneous group of circulatory conditions which if taken by

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themselves and without further test would be expected to give us a high mortality. We use the term "circulatory impairments" in a very broad sense and include not only heart murmur, hypertrophy, high and low blood pressures, pulses which are abnormal in rate and rhythm, but also certain conditions which, while not perhaps definitely circulatory in themselves, are associated with a high mortality from circulatory disease.

It should be appreciated that these tests are all made by medical men carefully chosen and trained for that purpose and that each examination was made "with special reference to the condition of the heart and circulation including a cardio-respiratory test" so that this is a selected group in so far that the examination can be accepted as of the highest grade which it is possible for us to obtain.

Among the large number of examinations that were made we find that of the 1220 cases involving the pulse there were

Tachycardia	780 or 63.93%
Bradycardia	64 or 5.25%
Intermittent	72 or 5.90%

while the irregular pulses were divided into two groups—first

Extra-systole	183 or 15.00%
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and in each of these cases the definite diagnosis of extra-systole was made by the examiner. We also include another group of

Irregular pulses	121 or 9.92%
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which were not otherwise classified by the examiner.

### TACHYCARDIA

Of this group of 780 cases there was no other impairment in 148 or 18.97%; in 82 or 10.51% it was associated with a high blood pressure; in 479 or 61.41% heart murmur was found and in 71 or 9.10% there was a goitre.

For the purposes of this discussion I have divided the reactions upon which our decisions were based, into "normal," "hypoactive," "hyperactive," "myocardial" (as representing a giving way of the systolic pressure apparently the result of a weakened myocardium) and the general term "unsatisfactory" in which class we have included a very considerable number of cases where the reaction was atypical but not easy to describe.

In the group where no other impairment was found we obtained a

Normal reaction	46.62%
Hypoactive	14.19%
Hyperactive	19.59%
Myocardial	6.08%
Unsatisfactory	13.51%

Where there was also a high blood pressure we find

Normal	23.17%
Hypoactive	00.00%
Hyperactive	48.78%
Myocardial	12.20%
Unsatisfactory	15.85%

with a heart murmur—

Normal	42.80%
Hypoactive	16.28%
Hyperactive	21.92%
Myocardial	15.45%
Unsatisfactory	3.55%

with goitre—

Normal	49.30%
Hypoactive	16.90%
Hyperactive	4.23%
Myocardial	15.49%
Unsatisfactory	14.08%

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In all of these cases the decision as to the declination or acceptance of the risk was based on the result of the cardio-respiratory test.

Of the group in which the rapid pulse was found alone we declined 42.57%; where it was associated with high blood pressure, 60.97%; with heart murmur, 49.27%; with goitre, 45.07%; and the total percentage declined of all of the tachycardias was 48.85%. The influence of age was not very marked—varying from 44.09% between 30 and 40 to 66.67% when the applicant was 60 or over.

### BRADYCARDIA

This group was a small one with 64 cases or 5.25% of the whole. There was no other circulatory impairment in 25 or 39.06%; there was a high blood pressure in 4 or 6.25%; heart murmurs in 33 or 51.56% and goitre in 2 or 3.13%.

The character of the reaction in those cases where the slow pulse stood alone was

Normal	44.00%
Hypoactive	28.00%
Hyperactive	4.00%
Myocardial	4.00%
Unsatisfactory	20.00%
with a high blood pressure—	
Normal	50.00%
Hypoactive	00.00%
Hyperactive	25.00%
Myocardial	00.00%
Unsatisfactory	25.00%
with a heart murmur—	
Normal	51.51%
Hypoactive	12.12%
Hyperactive	9.09%
Myocardial	21.21%
Unsatisfactory	6.06%

with a goitre there were only two cases, one of which gave a normal reaction and one hypoactive.

Of the entire group of bradycardia we declined 45.31%. Where it was the only impairment we declined 44.00%; when, with a high blood pressure 50.00%; heart murmur 45.45% and with goitre 50.00%.

In this group, there seems to have been a gradually increasing percentage of abnormal reactions with advancing years varying from 33.33% when, under 30 to 66.67% between 50 and 60; at 60 and over we declined 50.00%.

#### INTERMITTENT PULSE

Of this small group of 72 cases there was no other impairment in 46 or 63.89%; in 4 or 5.56% it was associated with a high blood pressure; in 22 or 30.56% heart murmur was found and there were no cases associated with goitre.

In this group where no other impairment was found, we obtained a

Normal reaction	52.17%
Hypoactive	17.39%
Hyperactive	2.17%
Myocardial	8.70%
Unsatisfactory	19.57%

Where there was a high blood pressure, we find

Normal	25.00%
Hypoactive	25.00%
Hyperactive	25.00%
Myocardial	00.00%
Unsatisfactory	25.00%

with a heart murmur

Normal	36.36%
Hypoactive	00.00%
Hyperactive	13.64%
Myocardial	36.36%
Unsatisfactory	13.64%

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Of the group in which the intermittent pulse was found alone, we declined 43.48%; where it was associated with high blood pressure 75.00%; with heart murmur, 22.00% and the total percentage declined of all intermittent pulses was 51.39%.

In this group, there seemed to be a very definite increase in the percentage of declinations in late life after 50, the percentage varying from 30.77% in 40-50 to 75.00% at 60 or over.

### EXTRA SYSTOLE

As has already been stated, this group is made up of cases in which the irregularity was, according to the examiner, definitely due to extra systole. It consisted of 183 cases or 15.00% of the whole. Of this group in 114 or 62.30% there was no other impairment; 9 or 4.92% it was associated with high blood pressure; 55 or 30.05% with a heart murmur and in 5 or 2.73% a goitre was also found.

The character of the reaction in those cases where the extra systole stood alone

Normal	66.67%
Hypoactive	14.04%
Hyperactive	4.39%
Myocardial	2.63%
Unsatisfactory	12.28%

with a high blood pressure

Normal	33.33%
Hypoactive	00.00%
Hyperactive	22.22%
Myocardial	00.00%
Unsatisfactory	44.44%



## with a heart murmur

Normal	45.45%
Hypoactive	14.55%
Hyperactive	18.18%
Myocardial	16.36%
Unsatisfactory	5.45%

## with a goitre

Normal	80.00%
Hypoactive	20.00%
Hyperactive	00.00%
Myocardial	00.00%
Unsatisfactory	00.00%

Where the extra systole was found alone we declined 26.32%; where it was associated with high blood pressure—66.67%; with heart murmur—47.27%; there were no cases associated with goitre declined. The total percentage of all the extra systole declined was 33.89%.

Among these cases the relation to age was quite definite and unsatisfactory reactions to the test increased in frequency directly as did the age, varying from 15.63% between 20-30 to 100% at age 60 or over.

## IRREGULAR PULSE

Of this group of 121 cases there was no other impairment in 67 or 55.37%; in 8 or 6.61% it was associated with high blood pressure; in 43 or 35.54% with heart murmur and in 3 or 2.48% with goitre.

In this group where no other impairment was found, we obtained a

Normal reaction	46.27%
Hypoactive	22.38%
Hyperactive	5.97%
Myocardial	7.46%
Unsatisfactory	17.91%

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with high blood pressure

Normal	00.00%
Hypoactive	25.00%
Hyperactive	12.50%
Myocardial	25.00%
Unsatisfactory	37.50%

with a heart murmur

Normal	11.63%
Hypoactive	34.88%
Hyperactive	23.26%
Myocardial	27.91%
Unsatisfactory	2.33%

with a goitre

Normal	33.33%
Hypoactive	33.33%
Hyperactive	00.00%
Myocardial	33.33%
Unsatisfactory	00.00%

In this group in which the irregular pulse was found alone, we declined 50.75% ; where it was associated with high blood pressure, 100.00% ; with heart murmur, 81.40% ; with goitre, 66.67% and the total percentage of all irregular pulses declined was 65.29%.

In this group also there seemed to be a distinct increase in the percentage of declined cases associated with the older ages varying from 33.33% under 20 to 85.71% in those between 50 and 60.

The first impression made upon my mind by this investigation was the surprisingly large number of cases in which a very careful examination disclosed some other circulatory impairment than that on account of which the examination was demanded.

We find a single impairment of

Tachycardia	in	148
Bradycardia	"	25
Intermittent	"	72
Extra Systole	"	114
Irregular	"	67

a total of 426—34.92% of the whole group where the pulse impairment was the only one discovered.

Where Tachycardia stood alone we declined 42.57%

Bradycardia	"	44.00
Intermittent	"	26.32
Extra Systole	"	50.75
Irregular	"	43.48

Where the pulse impairment was combined with high blood pressure we declined 64.49%; where it was combined with heart murmur 51.58%; where it was combined with goitre 43.21%.

Of this total number (1,220) cases in which the cardio-respiratory test was applied on account of the condition of the pulse we declined 588 or 48.20%.

If the cardio-respiratory test as developed by Frost can be accepted as a satisfactory measure of circulatory efficiency, and our experience during the last five years would apparently justify such a conclusion, we must accept the idea that those impairments of the circulation which are associated with irregularities in rate and rhythm of the pulse are in many instances manifestations of much more serious underlying impairments.

For a good many years we, of the New England Mutual have been satisfied that the attempts of many authors to subdivide and give names to various circulatory conditions and to assume that, having given it a name, they had full knowledge of the facts, was wrong in principle and that much of

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the difficulty which has obtained in controlling circulatory disease has depended upon this method of approaching the problem.

We are more and more convinced that heart murmurs, variations in blood pressures and pulse were simply different manifestations of an underlying and more fundamental condition and our experience with this test apparently justifies that conclusion.

No attempt has been made to work out the exact mortality which has resulted in the cases which are here reported as accepted as a result of this test simply for the reason that during the past five years there has been but one claim among these 632 cases, and it may be interesting to know that that especial case was a comparatively young man who had been seen by a first-class medical man in his home town and a consultant in Boston, both of whom had told him that his whole trouble was a few extra systoles and, therefore, unimportant. He gave us a perfectly satisfactory cardio-respiratory test and a moderate amount of insurance was issued. He died suddenly within two months when apparently in good health. No autopsy was made, but it is generally assumed that death was due to a sclerosis of the coronary artery.

Dr. Hobbs—Dr. Cook.

Dr. Cook—Mr. President and Gentlemen: Doctors Patton and Mackenzie have given us a very scholarly as well as practical presentation of the subject, and have so fully covered it, with such a detailed review of the authoritative literature, that an expression of appreciation would fill the usual requirement of discussion.

I feel that it was a wise choice of subjects by the program committee which included pulse rates and irregularities, as unquestionably we have been guilty of neglecting this important sign of cardiac and general health, especially in this country. Our literature has been concerned more with auscultation

tory findings and graphic records, while the English and French clinicians have laid on the pulse the emphasis the subject justified, especially Mackenzie, Lewis, Huchard, and Vaquez.

In twenty-five years' experience in reviewing medical examination blanks, I recall very few instances in which an examiner did more than note an irregularity or intermission and counted the number per minute. The average examiner does not, without prompting and re-examination, furnish sufficient evidence on which to exclude heart block and other serious abnormalities. In fact, irregularities are so infrequently reported that I am of the opinion that probably only the more serious ones come to our attention. For example, respiratory irregularity is very seldom reported, and, considering the number of young applicants in which sinus irregularity is probably present, and the infrequency with which it is mentioned in an insurance examination, the average examiner must either overlook it or regard it as not worthy of mention.

Sir James Mackenzie is a very eminent clinician, but I would not feel safe in having him pass upon applicants for life insurance with a pulse abnormality. He says, "In some remote past the idea arose that a heart to be normal must be regular in its rhythm, and that any irregularity must be looked on with suspicion. A few years ago when I was present at a meeting of medical directors for life insurance, a body reckoned to consist of specialists on prognosis, I was struck by their attitude towards irregular heart action. Experienced physicians stated that they systematically penalized or rejected candidates if the heart showed any form of irregularity." He further states, "Extra systoles are absolutely no cause for alarm; they are of no more significance than gray hairs."

This is the extreme position of the propagandist who has

recognized that physicians had been unnecessarily alarmed on the discovery of an irregular pulse in young people—the so-called respiratory or sinus irregularity—and in his effort to combat this fallacy, he has become much too lenient and has probably been led into an equally mistaken position.

I believe that the contrary statement of Lewis is more apt to be correct—that “All departures from the normal mechanism of the heart should call for special investigation into the fitness of the heart as a whole”; and we could add, into the fitness of the body as a whole—for many of these pulse departures may be extracardial in origin but none the less serious, e. g., goitre and hypertension.

In considering Dr. Patton's and Dr. Mackenzie's summary paragraph by paragraph:

1. *The exercise test.* I agree that, in spite of the criticism of some eminent internists, the exercise test can assist us in forming a judgment of cardiac competence and of the significance of pulse abnormalities. We should, I believe, lay greater stress on any symptoms of dyspnoea or discomfort or pain which develop as a result of exercise, than most of us have in the past. We have relatively overemphasized the effect on pulse rate, and although this should be noted and considered, I think we can be seriously misled if we become too dogmatic or mechanical in the interpretation of the exercise test. Mackenzie states that the pulse of a trained athlete returns to normal rate after exercise more slowly than in the sedentary, and that the response to effort in myocardial conditions does not produce a greater increase in rate than in healthy hearts. We must also keep in mind Lewis' statement, that exercise restores the normal rate in partial heart block.

The danger in devising any mechanical test is that we gradually come to depend on it alone—because it is definite, clear cut, and furnishes us with a ready, though deceptive, substitute for judgment. I believe this is why men like Mac-

kenzie and Vaquez declaim so against tests. They know medicine is never an exact science, and that cardiac capacity—probably the most complicated of all medical problems—cannot be measured off like yards of cloth. Therefore, rather than have us fall into a more grievous sin, they would deprive us of what little help the device may contribute. However, we shall probably retain the exercise test in insurance work, and guard against it becoming a fetish as best we may.

2. In the second paragraph of the summary, I think we all agree without comment that paroxysmal tachycardia, auricular flutter and fibrillation, heart block, and pulsus alternans, are uninsurable.

3. The third statement is, I feel, too broad for acceptance, and broader than the essayists really mean—"that true bradycardia, true tachycardia, when neither is the result of impairment local\* or remote, are insurable at standard rates." In the first place, this statement imposes no limit of slowness or rapidity. I think most of us will prefer to maintain the lower limit of 50, which, in the course of the paper, is mentioned as the practice of the Prudential. A moderately slow pulse has been shown to be a favorable sign, but when we consider pulse rates in the 40's or less, I believe most of us will think of the too likely possibility of heart block or hypertension to trust an examiner to exclude serious pathology. The same is true in rates above 90. The essayists here emphasize the importance of excluding Graves' disease, tuberculosis, asthenias, and heart disease; but they do not tell us and I do not know how this can be done in the ordinary insurance examination, nor do they caution in regard to the gravity of the higher pulse rates at older ages as evidence of some serious toxic respiratory or especially circulatory disturbance. I personally believe that a pulse rate over 90 above age forty calls for very serious consideration.

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4. The fourth paragraph of the summary, I believe, is of great importance in tending to correct the mistaken impression that some eminent teachers have given in regard to the inconsequential nature of extra systoles. Doctors Patton and Mackenzie state that over 50 they give rise to grave doubt as to the myocardium being normal. I go further, in the opinion that over age 40 extra systoles of sufficient frequency to be caught in one-fourth to one-half minute insurance examination pulse count, should be regarded with grave concern. It is probably true that everyone has at some time an occasional extra systole, but that is very different from two, three, five, or more every minute in an insurance examination. Lewis states: "Among patients admitted to dispensaries and hospitals, premature contractions are most common in those who exhibit definite signs and symptoms of cardiac disease. Of the factors which appear to be predominantly associated with the gross lesions of the heart, premature contractions stand first." Vaquez agrees with Huchard that "any arrhythmia at or after middle age should be investigated. Often it is the first indication of hypertension, announcing the eventual occurrence of failure of left ventricle."

While infrequent extra systoles may, in young adults, be safely taken with rigid selection—and an experience obtained no higher than the 113% of the Medico-Actuarial investigation—I believe that the majority of persisting abnormalities of rate and rhythm in cases over thirty-five years of age, are due to some progressive cardiovascular or other serious change which will definitely affect the longevity of this group.

Again I want to thank Doctors Patton and Mackenzie for their stimulating, exhaustive, and interesting presentation of this subject.

Dr. Hobbs—Dr. Wishard.

Dr. Wishard—Mr. President and Gentlemen: I appreciate



that you are all quite weary and prefer not having a long-winded article at this time. A few things, however, that I would care to say may be a repetition of some things that have been said. You appreciate my difficulty in attempting to say something at this time after so many things have been said in reference to the "Pulse in Life Insurance."

The thorough and scientific manner in which this subject has been dealt with by the authors deserves our highest commendation and offers a challenging appeal to us to devote more careful study to cardiac diseases.

Since deaths from heart impairments have for some time held first place in mortality experience, we, as medical directors, should give increasing attention to ways and means of detecting through life insurance examinations the potential as well as the actual heart cases which should be eliminated as undesirable risks.

The authors of this excellent paper have limited their discussion to four types of cardiac irregularities that are within the limits of acceptance when the symptoms are properly evaluated. These abnormalities are manifested by pulse readings distinctly atypical or at least deviating from the normal sufficiently to suggest further investigation to determine if possible the causative factor or factors.

Tachycardia, bradycardia, extrasystoles, and sinus arrhythmia, particularly the first three named conditions may be the expression of either a diseased myocardium or only a functional heart disturbance. Our real problem, therefore, and it is a very real one, is to secure enough data from the field examiner to enable us to arrive at proper conclusions.

The interpretations of field examiners of the significance of an abnormal pulse necessarily vary greatly depending upon their training and experience, and we, of the medical department, often have very little to guide us in passing judg-

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ment upon an abnormal pulse such as mentioned in this paper except the mere statement of the fact of its existence. It, therefore, should, I believe, become routine practice to require a functional heart test as suggested by Drs. Patton and MacKenzie or some other trustworthy test which will throw a definite strain upon the heart, and have the examiner record carefully the heart response, when dealing with an abnormally slow or fast pulse of obscure origin or where there are extrasystoles.

Our attention is called to the great necessity of adjusting properly the exercise heart test to the subject. This requires painstaking thought. It goes without saying that the functional heart test must be simple both in its application and interpretation. It is obvious that the unstable heart of a youth is likely to react to any given test more noticeably than is that of a more mature person, therefore, the test is more easily misinterpreted in the younger age group. War experience has demonstrated the unstable nervous control of heart rates in healthy young men, especially in students and those not much accustomed to manual labor. Furthermore, the functional heart test to admit of proper evaluation must take into full consideration the customary physical strain entailed by the applicant's daily routine. A diseased or potentially diseased heart may accustom itself to a certain amount of physical strain for a time without showing well defined symptoms of cardiac impairment. Thus, an individual with a slightly diseased myocardium may not show abnormal response to a prescribed functional heart test unless carried beyond the average test. Briefly, therefore, the test as emphasized by the authors must vary with the individual and must put a definite strain upon the heart muscle to be of distinct value. In making the test, the observer should weigh carefully all past unfavorable history which can be secured from the applicant such as recent illness, possibly of apparent-

ly minor significance, and careful investigation should be made to discover evidence of past or present focal infection.

It must be remembered that past performance of the heart under all conditions must be weighed in connection with the present ability of this organ to respond satisfactorily to a given heart test of whatever nature which puts a real strain upon this organ. The absence of distinct symptoms of cardiac embarrassment, or the improvement or disappearance of the symptom which suggests the necessity for a functional test, following such a test, is the best evidence that the heart probably is not seriously impaired.

Of the four types of pulse irregularities under discussion which permit favorable action under certain conditions, tachycardia and extrasystoles appear to deserve the most consideration.

The examiner should be able to differentiate readily between the history of a paroxysmal and non-paroxysmal tachycardia and report accordingly. The former, as we all know, begins abruptly with rapid acceleration of the heart which is very noticeable to the individual and the rate may reach 200 or more. The pulse may become so feeble that it cannot be counted.

The tachycardia of Basedow's Disease is, as we all recognize, not uncommon and may be confused with emotional tachycardia. The association of characteristic tremor should cause one to suspect hyperthyroidism. The pulse is usually small and regular and in the early cases may not exceed 100 to 120.

The rapid pulse of pure fright, apprehension or nervousness, is frequently encountered and is not always easily or at least properly differentiated from rapid pulse due to toxic causes such as tobacco, coffee, toxic goiter, and early tuberculosis without pyrexia. There is, in fact, frequently an as-

sociation of two or more of the above-named factors operating to produce an accelerated pulse beyond the usual limit of acceptance, especially at standard rates.

The tachycardia of indigestion and flatulency frequently occurs in middle life and sometimes earlier, especially in the neurotic and anemic types of individuals and those who also overindulge in tobacco, likewise, in tea or coffee.

Much significance should be attached to the tachycardia associated with hypertension. It suggests that the heart is experiencing much difficulty in overcoming peripheral resistance and any recent history of rapid heart action in a hypertension case should make one doubtful of accepting the risk even at very definite rating.

Extrasystoles represent the most frequent form of arrhythmias and, as stated by the authors, may be a symptom of almost all, if not, indeed, all heart diseases. They are due, it is claimed, to a disorder of the excitability of the myocardium. Thus abnormal strain on the heart may manifest itself in extrasystoles. If the threshold of excitability of the heart muscle is lowered from any cause, extrasystoles may result. The subjective symptoms may be very disquieting and annoying and out of proportion to the seriousness of the situation. The individual who is seldom conscious of extrasystoles may have a more greatly damaged myocardium than the one who is oversensitive to the disturbance. A slow extrasystolic pulse or bigeminal pulse may be due to a bradycardia in which instance the supplementary heart contraction does not reach the radial pulse. The rate may be as low as 40 pulse waves per minute. This pulse may be readily distinguished by putting the applicant through mild exercise such as walking about the room or by an injection of atropine, either of which is likely to change the pulse promptly.

It is noteworthy that certain drugs, especially digitalis and sodium salicylate, may produce extrasystoles and should be

thought of when other causes are undiscoverable. It must be remembered that extrasystoles occurring in those past fifty frequently indicate latent, if not present, arterial hypertension which alone might appear rather harmless but which may result in eventual failure of the left ventricle. On the other hand, they may be due to gastro-intestinal disorders the result of errors in diet or too rapid or too hearty eating; also to fatigue, grief, worry or overtaxing of the nervous system, all of which might yield readily to appropriate treatment. Excessive use of tobacco, coffee or tea and insomnia are all factors to be considered in connection with extrasystoles. It is obvious that many cases having an intermittent pulse are insurable but may be so only after a period of postponement during which time the cause or causes of the extrasystoles have been discovered and eliminated.

Bradycardia presents an interesting study, but since it is uncommon practice to accept pulse rates under 50 per minute, this condition does not give us a great deal of concern. A bradycardia that will respond satisfactorily to exercise or an injection of atropine should, as a rule, not worry us, especially where there is no past discoverable, unfavorable history or evidence of vertigo, syncope or other subjective symptoms of embarrassed heart action.

Cases presenting evidence of disturbed conduction of impulses (heart block) which do not respond satisfactorily to the exercise test or atropine are hazardous risks.

In dealing with persistent slow pulse between 50 and 60 per minute, we should endeavor to learn if this has been, so far as known, the average pulse throughout adulthood. Also, it should not be forgotten that syphilis may be the forerunner and essential cause of a bradycardia as it is, likewise, of a number of other heart conditions.

Sinus aorhythmia needs no further discussion as it is readily recognized and its significance is fully appreciated.

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In conclusion, permit me to say that the conviction is forced upon me that there is no question involving medical selection comparable with the one which has to deal with the ability of the heart to carry the individual to three score years and ten or beyond. We spend much time and earnest thought on the technique of urinalysis in order to detect more accurately the quantitative amounts of albumen or sugar, as the case may be, in an applicant's urine, often, I fear, forgetting that these undesirable urine findings may only be temporary and purely function in significance; but I raise the question: are we giving serious enough thought to cardiac problems which, in the aggregate, mean far more in their relation to mortality statistics? My plea is that we study the pulse more seriously as a barometer of heart efficiency and become better able to assist the field examiner who, in the stress of his professional activities too often has little time and frequently less disposition to become expert in cardiac interpretations.

Dr. Hobbs—Dr. Martin.

Dr. Martin—Mr. President and Gentlemen: Perhaps I may be pardoned instead of giving the prepared remarks I had in mind, if I may start more or less *de novo* in view of all all we have already heard from the men who so admirably dealt with the papers on murmurs and on "The Pulse Rate." I want to congratulate Dr. Patton and Dr. MacKenzie, as well as the others who have contributed to this discussion. I think they have left little further to be added.

I do not think that the papers of Dr. Patton and Dr. MacKenzie quite designate the full significance of the subject. It is quite obvious that when they speak of the pulse in relation to life insurance that they are really speaking of the whole vascular system and it is equally obvious that they require, in connection with the consideration of the pulse, an estimate of all the other factors possible in connection with

the circulatory system. Now, it strikes one as a clinician rather than an expert on insurance, that that is really the crux of the whole situation. We cannot estimate by any one means the functional efficiency of the heart. Sir James MacKenzie used to say, as we all know, that the history of the patient and his response to effort in the ordinary routine of his daily life is a very good guide to the heart's efficiency. On the other hand, we do know that there are very many types of cardiac disease in which the response to effort is excellent and yet these hearts are very seriously affected. I need only refer to the cases of aortic disease with insufficiency—in which very often the response to effort is normal. Many cases of such are on record. I need only refer to cases such as were described by Dr. Dwight of coronary disease, to the many cases of angina pectoris, all of which may at times give a normal response to effort if the ordinary tests are employed; it is for that reason one would think, as a clinician, that it is far more important not to depend on any one test if possible but to combine every feature of the vascular system to get an accurate estimate of what the heart is likely to do in the future—and that is not easy. Of course, for the average medical examiners, that is not easy. The suggestion of Dr. Cook is excellent; there should be some committee of you, gentlemen, who are experts in life insurance work to frame suggestions of universal application, and that may contribute in a research way to our knowledge of this subject. I don't think any university could ever contribute to our knowledge of efficiency of the heart in the efficient way that life insurance organizations such as this can do. The field is enormous, the opportunities of estimating the prognosis are far greater with insurance experts than they are with the average clinician who sees only a comparatively small number of cases, and for that reason I do believe that the field for

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information in this line that will be of benefit to the community in general is quite inestimable.

So far as the effort tolerance tests are concerned, may I say this while it is true that there are many cases in which they afford no information, nevertheless, either one of the two tests mentioned will, in a large majority, give information which is of distinct use. On the other hand, the number of conditions which affect the value of that test are also not appreciated, I think, by the average field examiner. I mean by that, constitutional defects, previous illnesses and the other things mentioned by so many before. My own point of view of the great feature of the effort tolerance test is the negative value. When an individual responds well to a test, one may conclude with a certain amount of reason that the myocardium is normal; I can recall many exceptions, one being a rather serious and important insurance risk. I only learned afterwards that it was an insurance case, but I saw in my ordinary routine, a man who had led an active life for 52 years, very active, and who was a bit of an athlete in his way and had heavy business responsibilities. That man had never been ill before, but at 52 years of age he ran for a street car, or at any rate in running across the street, exerted himself and immediately afterwards had his first attack of serious illness, from which he died ten days later. The autopsy showed an aneurism of the heart due to a serpigenous ulcer of the ventricle at the apex. In another case of cardiac disease, in a man who equally had led an active life, the electrocardiogram was the one and only thing that showed a definite lesion. That man died suddenly a day or two afterwards. He had been an active man in athletics. He had indulged in a great many active exercises and yet showed nothing to indicate that he had cardiac disease. Death was due to coronary thrombosis.

So one may find a great many exceptions to the rule in



these tests, so far as irregularities are concerned and I think we are all in accord with that view.

Regarding what has been said about the "ordinary arrhythmias" if one may use that term, I believe that a very careful study of the patient can alone determine whether or not there is a constitutional defect of no importance or whether or not there is behind it all some serious lesion of a latent kind which produces that attack. With reference to pulse rate, certainly everything over 100 per minute would lead me to suspect something more than a mere peculiarity of that individual.

I would like again to say how much I appreciate all these papers. I have learned a great deal here which has been of intense interest, and I think we should all be very grateful to the members who have prepared these contributions.

Dr. Hobbs—Does anyone wish to speak on the subject under consideration? Now, we will ask Dr. Patton to close the discussion.

Dr. Patton—I want to say one or two words of thanks to those who have discussed the paper. We believe that to be a pretty fair cross section of the opinion of the Association. We did not expect, in presenting the pulse, to indicate that the pulse was the only thing we were after. It is, as Dr. Martin said, the representation of one of the effects on the vascular system, one most readily observable by the average examiner. While we are very ready to accept Dr. Dwight's statement in regard to his cardio-respiratory test, he himself says that it has to be in the hands of a trained examiner as yet. What we want is the best representation we can get from the average field examiner. We must have, therefore, a simple test which the average examiner can apply. So far as these are concerned, the simple exercise test is the best we have to date for that purpose. We are very willing and ready to change to something else that the average examiner

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can use whenever we have something brought to our attention which is better than that. So far we haven't seen it.

There are several features that came up in connection with the discussion, but our idea is to get what we can, as an index of myocardial condition. We believe it isn't the murmur in itself or the rapid pulse in itself; it is what we can get as an indication of the true condition of the individual that is examined. We do not care what his age is and what his station in life. We are up against the proposition of having to apply some form of test that can be very generally applied by the average examiner.

Dr. Hobbs—As to the advisability of appointing a committee, the committee should come from some district where they could meet and where there could be an interchange of views. I will pass this idea along to my successor, Dr. Beckett. I have one sin of omission to confess, that yesterday morning I forgot to ask you, gentlemen, who had any questions to ask to put them on paper and they would be considered at the meeting today. However, you will remember that you did get a letter to this effect and it seems to me that if you had anything in your minds that you would take advantage of this. There are two or three things, however, which I know Dr. Weisse will speak to us about, having heard from some of the members of the Association. Dr. Weisse.

Dr. Weisse—Our President, Dr. Hobbs, asked me some time ago if I would be responsible for the answering of questions which would be sent to me in reply to his circular letter of June 4th. As a result of that circular letter I received five questions.

Dr. Turner, of the Jefferson Standard, wrote me with reference to the question of disabilities which will be answered in a paper from our Assistant Inspector of Risks, Mr. Austin D. Reiley.

Dr. Piper, Medical Director of the Guardian Life, raised a question with reference to the M. I. B. Code which I referred to Dr. Rogers, who is Chairman of the M. I. B. Committee, for reply.

Dr. Shewbrooks, Medical Director of the Lincoln National Life, asked a question with reference to reporting urinary impairments, which I referred to Dr. Patton, who is Chairman of the Urinary Committee, for answer.

Dr. Daniel, of the Great Southern Life Insurance Co. brought up the question of the danger point in systolic and diastolic blood pressure. I advised him, I thought it would be answered in the Dr. Rogers and Mr. Hunter paper on Blood Pressure.

Dr. VanDervoort, of the Fidelity Mutual Life, asked the question as to why the use of the green slip was discontinued in connection with examiners. I advised him that the green slip fell into general disuse over fifteen years ago, and that the reason for our Company discontinuing it was that we felt we did not get sufficient information to warrant the continuance of the procedure and that I thought that was the experience of many of the Companies.

#### DISABILITY BENEFITS SELECTION FROM THE UNDERWRITER'S STANDPOINT

BY AUSTIN D. REILEY

*Assistant Inspector of Risks, Mutual Life Insurance Company*

The first thing to be done in attacking this subject is to define somewhat the underwriter's standpoint. So many different angles of the problem must be considered by the underwriter in selecting his risk for disability benefits, that it is difficult to say what are the purely underwriting problems. From the wording of the request for this discussion, however, it would seem that what is desired is an analysis of the

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selection situation with particular attention to those classes in which malingering is easy and profitable. The subject will, therefore, be dealt with principally on that basis.

The first thing to do is to segregate the classes under discussion. Before doing that, however, it is well to call attention to one or two fundamentals. With rare exceptions, in the classes usually discussed in this connection, the chance of malingering is apparently obvious, and one thing that the underwriter has learned to distrust is the apparently obvious. Again, it is probable that the malingerer is constitutionally so inclined and will create his own opportunity even though it entails some loss to himself; and, even though heavy penalties are imposed on those classes where malingering seems easy, it will not by any means stop the effort to malingering.

The classes where malingering seems most easy would appear to be as follows:

1. Those insured up to and beyond their actual earning capacity.
2. Married and otherwise dependent women.
3. Children under self-supporting age.
4. Those not dependent on their personal efforts to gain a livelihood.

We will endeavor to examine each one of these classes separately.

In the first class, the question usually arises in case of the larger risks and is harder to handle as adverse decisions give rise to rather intense field irritation. It is, of course, true that the disability benefits clause is an indemnity strictly against loss of earning power and at first glance should be considered solely on that basis. There are, however, other factors which deserve consideration before we adopt hard and fast rules limiting benefit income insurance to certain definite proportions of earning capacity. Some of these fac-

tors are age, general character, type and surroundings, and chances of increase or decrease of earning power. What we should bear in mind is that in handling this problem we are endeavoring to keep out risks who possibly will attempt to malingering, and not to establish hard and fast rules which will lose us a good volume of business, which cannot be considered as a part of a class at all liable to malingering.

The factors I have mentioned are valuable in determining whether or not the applicant has come anywhere near reaching the peak of his earning power. From a purely common sense standpoint it seems hardly possible that any comparatively young man would voluntarily relinquish all future opportunities of a fine career for enforced complete inactivity and receipt of a very moderate income. It seems, therefore, that this is largely a problem that centers around the individual risk and can seldom be handled on the bases of rules and ratios.

I fear we are, all of us, too prone to forget that insurance must be sold; that our annual volume of business is produced by highly organized and expensive sales organizations, and that these organizations require arguments and talking points. It has always been considered a legitimate sales argument that insurance should be bought young, and that a possibly heavier burden in premiums should be carried when young in order to avail oneself of the smaller premiums. The argument that a risk should, while indemnifying his present earning capacity, also provide for a certain part of its natural increase, is far from a faulty argument. It is the underwriter's business to distinguish the risk in the case of which such procedure is dangerous.

But before we can consider the individual we must consider the class from which we are going to remove him if found worthy, and here, of course, certain standards are necessary. It is, of course, generally understood that the

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amount of disability benefits carried in other companies is just as vital a factor in selection as though it were all in the company to which the risk is applying. Again, the mere fact that the payment of premiums is provided for, as in corporation insurance, has no bearing on disability benefits selection. Very often in selecting business from the purely life insurance standpoint, we treat as entirely separate the personal insurance carried by the risk and that carried by a corporation for its own protection. It is a perfectly legitimate selling argument in inducing a corporation to insure a valued employee, that in case of disability they will have to pay him a portion of his salary, and can cover themselves against that loss by disability benefits insurance. Nevertheless, the disability benefits asked for in a corporation policy must be considered as part of the total income insured will receive in case of disability, and treated on that basis.

The standards adopted by the various companies, either tentatively or absolutely, vary greatly. At least one company has announced that it will not grant disability coverage beyond one-half of the risk's earned income, considering disability income insurance carried in all companies. From this the standards vary all the way to seventy-five per cent or more. Our own standard is about two-thirds.

It would seem that a fair standard which would consider all the factors necessarily involved would be about as follows:

1. For risks who have through age or other reasons reached about the peak of earning capacity, insure with disability benefits to one-half of earned income.
2. For risks who have the largest part of their earning power ahead of them, insure with disability benefits up to their full present earning capacity.
3. For risks that fall between these two extremes, insure with disability benefits up to two-thirds of present earning capacity.

Discussion will, of course, arise as to whether the proceeds of an established business to which applicant gives his full time and energy constitute earned income. Our instructions to our own Inspectors are that if the business depends for its success on the applicant's efforts, its proceeds are to be considered as earned income.

It would be futile to leave this part of our subject without some inquiry into the method of obtaining the information necessary to the selection outlined above.

Appended to this paper, labeled Exhibit A, will be found the Mutual Life's inquiry blank as to the amount of insurance with disability income coverage carried in all companies. This must be furnished in all cases where the disability benefits are asked for in excess of \$25,000 insurance. We may ask for it in other cases where the amount of insurance stated as carried in other companies is such that if it carries disability benefits the risk would be over-insured. It would be well if companies added to their application blanks a question as to the amount of insurance with disability benefits carried in other companies.

As to the information regarding earned income, we have in the main to depend on our inspection sources, and here one fact should be noted. Any underwriter who has studied inspections has noted that income is almost invariably understated. This gives us a slight margin of safety, if we hew closely to our inspection report in the mass of cases. Where inspection is made by salaried inspectors we know that in the great bulk of cases his information is conservatively correct. The difficulty arises principally in the case of reports made by correspondents, and in these we have to use our underwriting judgment in estimating earned income. But again, in the great mass of cases, I do not think we will go far wrong.

I have, of course, no statistical data to contribute as to

this class, and I am quite sure no other company has. Perhaps we may be able to get some light on the matter by examining our class of larger risks from the standpoint of disability benefits experience, but this we have not done as yet. I think we have all seen certain cases undoubtedly over-insured for benefits who have presented claims. In the majority of such claims that I have seen, the over-coverage was caused by evident lack of knowledge as to the amount of disability benefit coverage carried in other companies.

#### MARRIED AND OTHERWISE DEPENDENT WOMEN

Practically all companies appear to have had an adverse experience in insuring women for disability benefits, and in consequence a majority have very drastic rules for their selection. These rules seem to bear almost wholly on the class of married women. A recent examination of the actions of 21 of our esteemed competitors elicited the information that 14 did not insure dependent married women at all, and 11 of these took no married women. I think from the beginning of disability benefit insurance the tendency to exclude married women has been strong. This, of course, on account of the fact or opinion that in most instances they have no earned income. In our company we took the view that a married woman earned a definite part of her husband's income. I think that the largest class we all insure is farmers, and it is very certain that as regards farmers' wives the above statement regarding earnings is fact. Our usual practice on the smaller cases up to say \$10,000 of insurance, is to regard the wife as available for disability benefits, up to 1/4 of her husband's income. Whether our position in this is faulty or not can only be determined when sufficient experience has been gathered, which is not the case up to the present time. For purposes of present selection it is admitted that married women must be somewhat limited in amount. It



also appears that there are certain causes of disability that will probably show in a woman's disability experience table in excess of what will appear in a man's. One of these is tuberculosis.

Another point presents itself here. In general, there is not a great deal of restriction placed on single, self-supporting women for disability benefits, except as to amount. A few companies have special clauses reducing the value of the benefit, charging extra premium, or lengthening the 90-day provision; but in general, single, self-supporting women seem to be given this benefit freely. The following table shows the average number of women insured as spinsters who afterward marry:

1,000 Spinsters Selected at Random Who Were Insured in 1919 With the Number Existing at the End of 5 Years, Together With the Number of Applicants Who After Carrying Their Insurance for 5 Years Are Married

Ages at Issue	Spinsters Insured in 1919	Total Existing in 1924	Existing in 1924 Who Are Married	Percentage of Total Existing
15-29	754	581	249	42.9
30-44	205	184	25	13.6
45 & over	41	37	1	2.7
Total	1,000	802	275	34.3

From this it appears that 34% of the class we insure as spinsters will automatically transfer themselves to the class considered more unfavorable within five years of insurance.

From all of these considerations it would seem that the prime need in the selection of this class is the strict limitation of amount and a rigid operation of the rules of selection on impairments which we know tend toward the causes of high claim frequency, such as tuberculosis. The class fortunately is small, comprising only 2% of the total business on the amount basis, and we cannot be much hurt by carrying it along until we test out the power of our selection.

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We have not yet been able to secure real data on the effect of the ninety-day provision. I think we have all of us found that more careful selection was necessary as we increased the value of the clause, and we will expect to find the same necessity here both as regards men and women and, therefore, cannot form any definite conclusions until we see some real figures.

### **CHILDREN UNDER SELF-SUPPORTING AGE**

The third class we have to consider—children of dependent age—may be disposed of quite briefly. While apparently it possesses the opportunity to malingering, on close examination it is doubtful if this is true. While a boy of dependent age, say up to 21, may occasion no loss by disability at present, the effect on his future earning would undoubtedly be great. Surely no company would pay a disability claim on a boy who was attending school regularly. His education must therefore be interrupted at grave risk to his future. It may be said that in certain parts of the country this is not considered as important as in others. This is perhaps true of parts of Canada and the south, but the spread of compulsory education is fast and sure, and those parts of the United States that do not have it now will undoubtedly have it in the not very distant future.

As to the actual experience in this class, it is somewhat peculiar. With us, the experience under age 21 is better than from 21 to 29, and this is due almost entirely to the high frequency rate from tuberculosis. Below that age, that is, from 15 to 20, inclusive, the rate is not greatly in excess of the average, especially when taken by lives. The excess can be entirely accounted for by the high tuberculosis frequency. The class needs rigid amount restriction and also the usual selection methods which we follow to prevent speculation. We are also rather particular to see that this class measures up to a good environmental standard.

The last class is really too small for discussion. In busy work-a-day America we encounter few risks of disability benefit age who do not pursue some gainful occupation.

#### GENERAL SUMMARY

From the foregoing it will be seen that I am extremely doubtful whether there can be any class distribution of malingerers. While we do find an increased claim frequency in some classes where we might be inclined to suspect malingering, we can usually find causes quite as potent to account for it.

When we consider the large proportion of our applicants who apply for disability benefits we should gain sufficient experience soon to arrive at some idea of the effect of malingering in suspected classes by process of elimination.

Unfortunately, up to the present time, work in this direction has been interfered with by changes in the clause itself. In our own case much of the work we have done, under the yearly and monthly clauses, has been nullified by the adoption of the ninety-day provision. In the meantime we can only follow such leads as I have endeavored to indicate.

#### *Exhibit A*

### THE MUTUAL LIFE INSURANCE COMPANY OF NEW YORK

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*Special questions to be answered when Disability Benefits are  
desired in excess of \$25,000, including Old Insurance.*

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WHEN THIS BLANK IS NOT FURNISHED IT WILL BE ASSUMED  
THAT DISABILITY BENEFITS ARE DESIRED  
UP TO \$25,000 ONLY.

1—State the total amount of life insurance providing for

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**Disability Income** you now carry in this and all other companies.

Total Am't Ins. ....

Ann'l Dis. Income .....

2—Are you at present applying to other companies for insurance carrying the Disability Benefit feature? If so, state total amount and income.

Total Am't Ins. ....

Ann'l Dis. Income .....

3—State the total amount of weekly, monthly or annual income you would receive in addition to amount stated under Question #1 under all your health and accident insurance in the event of illness or disability.

Am't Income .....

(State whether weekly, monthly or annually)

Date \_\_\_\_\_

Applicant

.....  
**Witness**

Form 3382-50M-2-24

Ed. Feb.-1924

Dr. Hobbs—We thank Dr. Weisse and Mr. Reiley for their remarks. Mr. Secretary, is there any more business? Apparently, then, we have come to the close of our program and the time comes to adjourn until our next annual meeting. I wish to thank you all for your generous attendance and for your hearty co-operation, and I trust that next year may be full of good results and that some of the good results may come from the meeting which you have just attended.

The Annual Dinner of the Association was held at the Waldorf-Astoria on the evening of Tuesday, October 21st, 1926.

# LIST OF MEMBERS OF THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS.

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Walter A. Reiter, M. D.	Mutual Benefit, Newark, N. J.

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Frank P. Righter, M. D.	Atlantic Life, Richmond, Va.
J. A. Roberts, M. D.	Canada Life, Toronto, Ont.
A. J. Robinson, M. D.	Connecticut General, Hartford, Conn.
Thomas H. Rockwell, M. D.	Equitable, New York, N. Y.
Oscar H. Rogers, M. D.	New York Life, New York, N. Y.
Fred W. Rolph, M. D.	Confederation, Toronto, Ont.
Edward K. Root, M. D.	Aetna, Hartford, Conn.
Robert L. Rowley, M. D.	Phoenix Mutual, Hart- ford, Conn.
Charles L. Rudasell, M. D.	Life Ins. Co. of Virginia, Richmond, Va.
Eugene F. Russell, M. D.	Mutual, New York, N. Y.
H. Crawford Scadding, M. D.	Canada Life, Toronto, Ont.
C. E. Schilling, M. D.	Ohio State, Columbus, Ohio.
Samuel B. Scholz, Jr., M. D.	Massachusetts Mutual, Springfield, Mass.
Albert Seaton, M. D.	American Central, Indi- anapolis, Ind.
George H. Shaw, M. D.	Travelers, Hartford, Conn.
Joyce T. Sheridan, M. D.	Philadelphia Life, Phila- delphia, Pa.
Arthur L. Sherrill, M. D.	Equitable, New York, N. Y.
Daniel M. Shewbrooks, M. D.	Lincoln National, Fort Wayne, Ind.

## List of Members

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Joseph L. Siner, M. D.	Fidelity Mutual, Philadelphia, Pa.
Donald W. Skeel, M. D.	Occidental, Los Angeles, Calif.
Dewitt Smith, M. D.	Southwestern, Dallas, Texas.
James M. Smith, M. D.	American Central, Indianapolis, Ind.
Malcolm K. Smith, M. D.	Prudential, Newark, N. J.
Wm. B. Smith, M. D.	Connecticut General, Hartford, Conn.
Thayer A. Smith	Mutual Benefit, Newark N. J.
Morton Snow, M. D.	Massachusetts Mutual, Springfield, Mass.
Marion Souchon, M. D.	Pan-American, New Orleans, La.
Howard B. Speer, M. D.	Metropolitan, New York, N. Y.
Samuel C. Stanton, M. D.	Farmers National, Chicago, Ill.
Henry F. Starr, M. D.	The Pilot, Greensboro, N. C.
John B. Steele, M. D.	Volunteer State Life, Chattanooga, Tenn.
S. J. Streight, M. D.	Canada Life, Toronto, Ont.
Carl Stutsman, M. D.	Merchants Life, Des Moines, Iowa.
Lawrence G. Sykes, M. D.	Connecticut General, Hartford, Conn.
William Thorndike, M. D.	Northwestern Mutual, Milwaukee, Wis.
Walter E. Thornton, M. D.	Lincoln National, Fort Wayne, Ind.

### 308      Thirty-Seventh Annual Meeting

Paul E. Tiemann, M. D.	New York Life, New York, N. Y.
Harry Toulmin, M. D.	Penn Mutual, Philadelphia, Pa.
Frank L. Truitt, M. D.	Reserve Loan, Indianapolis, Ind.
John S. Turner, M. D.	Southland, Dallas, Texas.
Joseph P. Turner, M. D.	Jefferson Standard, Greensboro, N. C.
Henry G. Tuttle, M. D.	Metropolitan, New York, N. Y.
Charles A. Van Dervoort, M. D.	Fidelity Mutual, Philadelphia, Pa.
Euen Van Kleeck, M. D.	Travelers, Hartford, Conn.
George A. Van Wagenen, M. D.	Mutual Benefit, Newark, N. J.
Albert A. Wagner, M. D.	Reliance Life, Pittsburgh, Pa.
Charles E. Waits, M. D.	Southern States, Atlanta, Ga.
William R. Ward, M. D.	Mutual Benefit, Newark, N. J.
William H. E. Wehner, M. D.	Fidelity Mutual, Philadelphia, Pa.
Faneuil S. Weisse, M. D.	Mutual, New York, N. Y.
Ernest A. Wells, M. D.	Aetna, Hartford, Conn.
Fred L. Wells, M. D.	Equitable of Iowa, Des Moines, Iowa.
David E. W. Wenstrand, M. D.	Northwestern Mutual, Milwaukee, Wis.
Charles D. Wheeler, M. D.	State Mutual, Worcester, Mass.



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Charles M. Whicher, M. D.	Royal Union, Des Moines, Iowa.
Chester F. S. Whitney, M. D.	Home Life, New York, N. Y.
Alfred A. Willander, M. D.	Mutual Trust, Chicago, Ill.
Thomas H. Willard, M. D.	Metropolitan, New York, N. Y.
Richard Lee Willis, M. D.	Mutual, New York, N. Y.
Charles H. Willits, M. D.	Provident Mutual, Philadelphia, Pa.
Edwin B. Wilson, M. D.	Mutual, New York, N. Y.
Gordon Wilson, M. D.	Maryland Life, Baltimore, Md.
McLeod C. Wilson, M. D.	Travelers, Hartford, Conn.
E. E. Wishard, M. D.	Public Savings, Indianapolis, Ind.
Harry P. Woley, M. D.	New York Life, New York, N. Y.
Glenn Wood, M. D.	Illinois Life, Chicago, Ill.
G. Elliott Woodford, M. D.	Connecticut General, Hartford, Conn.
Wade Wright, M. D.	Metropolitan, New York, N. Y.
John C. Young, M. D.	American Life, Detroit, Mich.

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**HONORARY MEMBERS**

John W. Brannan, M. D.	London, England
John K. Gore,	Newark, N. J.
Arthur Hunter	Hartford, Conn.
Thomas Glover Lyon, M. D.	New York, N. Y.
Edward E. Rhodes	New York, N. Y.
Archibald A. Welch	Newark, N. J.
Granville M. White, M. D.	New York, N. Y.

## COMPANIES AND THEIR REPRESENTATIVES

Aetna Life, Hartford, Conn.	{ D. B. Cragin, M. D. W. E. Dickerman, M. D. P. H. Ingalls, M. D. E. K. Root, M. D. E. A. Wells, M. D.
American Central Life, Indianapolis, Ind.	{ Albert Seaton, M. D. J. M. Smith, M. D.
American Life, Detroit, Mich.	J. C. Young, M. D.
Atlantic Life, Richmond, Va.	F. P. Righter, M. D.
Bankers Life, Des Moines, Iowa	{ Ross Huston, M. D. A. E. Johann, M. D.
Bankers Reserve Life, Omaha, Neb.	W. F. Milroy, M. D.
Berkshire Life, Pittsfield, Mass.	{ Henry Colt, M. D. B. W. Paddock, M. D.
Canada Life, Toronto, Ont., Can.	{ H. S. Hutchinson, M. D. J. A. Roberts, M. D. H. C. Scadding, M. D. S. J. Streight, M. D.
Capitol Life of Colorado, Denver, Colo.	{ J. W. Ames, M. D. George Atcheson, M. D.
Central Life of Illinois, Chicago, Ill.	T. W. Burrows, M. D.

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Central Life As. Soc. of U. S., Des Moines, Iowa	M. I. Olsen, M. D.
Columbia National Life, Boston, Mass.	{ F. R. Abbe, M. D. J. S. Phelps, M. D.
Commonwealth Life, Louisville, Ky.	W. F. Blackford, M. D.
Confederation Life Assn., Toronto, Ont., Can.	{ E. M. Henderson, M. D. F. W. Rolph, M. D.
Continental Assurance Co., Chicago, Ill.	H. W. Dingman, M. D.
Connecticut General Life, Hartford, Conn.	{ A. J. Robinson, M. D. W. B. Smith, M. D. L. G. Sykes, M. D. G. E. Woodford, M. D.
Connecticut Mutual Life, Hartford, Conn.	{ C. D. Alton, M. D. J. B. Hall, M. D. H. A. Martelle, M. D.
Equitable Life Assur. Soc., New York, N. Y.	{ A. W. Billing, M. D. F. G. Brathwaite, M. D. R. M. Daley, M. D. Arthur Geiringer, M. D. F. W. McSorley, M. D. H. L. Mann, M. D. H. S. Pearse, M. D. T. H. Rockwell, M. D. A. L. Sherrill, M. D.
Equitable Life of Iowa, Des Moines, Iowa	F. L. Wells, M. D.
Excelsior Life, Toronto, Ont., Can.	John Ferguson, M. D.

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Farmers National Life, Chicago, Ill.	S. C. Stanton, M. D.
Federal Life, Chicago, Ill.	F. L. B. Jenney, M. D.
Fidelity Mutual Life, Philadelphia, Pa.	{ J. L. Siner, M. D. C. A. Van Dervoort, M. D. W. H. E. Wehner, M. D.
Franklin Life, Springfield, Ill.	O. F. Maxon, M. D.
Great Southern Life, Houston, Texas	{ J. E. Daniel, M. D. R. M. Hargrove, M. D.
Great West Life Assur., Winnipeg, Man., Can.	{ R. J. Blanchard, M. D. W. L. Mann, M. D.
Guardian Life, New York, N. Y.	{ M. B. Bender, M. D. C. B. Piper, M. D.
Home Life, New York, N. Y.	{ F. W. Chapin, M. D. C. F. S. Whitney, M. D.
Illinois Life, Chicago, Ill.	Glenn Wood, M. D.
Imperial Life Assur., Toronto, Ont., Can.	{ H. B. Anderson, M. D. R. W. Mann, M. D.
Jefferson Standard Life, Greensboro, N. C.	{ J. T. J. Battle, M. D. J. P. Turner, M. D.
John Hancock Mutual Life, Boston, Mass.	{ E. H. Allen, M. D. W. B. Bartlett, M. D. Byam Hollings, M. D. R. A. Behrman, M. D.
Kansas City Life, Kansas City, Mo.	H. A. Baker, M. D.

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Life Ins. Co. of Va., Richmond, Va.	C. L. Rudasell, M. D.
Lincoln National Life, Fort Wayne, Ind.	{ C. H. English, M. D. D. M. Shewbrooks, M. D. W. E. Thornton, M. D.
London & Scott. Assur. Assn., Montreal, Que., Can.	A. H. Gordon, M. D.
London Life, London, Ont., Can.	Angus Graham, M. D.
Manhattan Life, New York, N. Y.	G. H. Barber, M. D.
Manufacturers Life, Toronto, Ont., Can.	T. F. McMahon, M. D.
Maryland Life, Baltimore, Md.	Gordon Wilson, M. D.
Massachusetts Mutual Life, Springfield, Mass.	{ L. D. Chapin, M. D. R. B. Ober, M. D. S. B. Scholz, Jr., M. D. Morton Snow, M. D.
Merchants Life, Des Moines, Iowa	Carl Stutsman, M. D.
Michigan Mutual Life, Detroit, Mich.	W. G. Hutchinson, M. D.
Midland Mutual Life, Columbus, Ohio.	Frank Harnden, M. D.
Minnesota Mutual Life, St. Paul, Minn.	C. N. McCloud, M. D.

# Companies and Representatives 315

Metropolitan Life,  
New York, N. Y.

J. L. Adams, M. D.  
C. L. Christiernin, M. D.  
J. N. Coolidge, M. D.  
E. M. Holden, M. D.  
J. C. Horan, M. D.  
A. O. Jimenis, M. D.  
R. J. Kissock, M. D.  
A. S. Knight, M. D.  
A. J. Lanza, M. D.  
W. S. Manners, M. D.  
S. W. Means, M. D.  
J. C. Medd, M. D.  
G. L. Megargee, M. D.  
J. B. Ogden, M. D.  
H. B. Speer, M. D.  
H. G. Tuttle, M. D.  
T. H. Willard, M. D.  
Wade Wright, M. D.

Missouri State Life,  
St. Louis, Mo.

B. Y. Jaudon, M. D.

Mutual Benefit Life,  
Newark, N. J.

C. D. Bennett, M. D.  
C. P. Clark, M. D.  
F. W. Hagney, M. D.  
Archibald Mercer, M. D.  
W. A. Reiter, M. D.  
T. A. Smith, M. D.  
G. A. Van Wagenen, M. D.  
W. R. Ward, M. D.

Mutual Life Assur. of Can.,  
Waterloo, Ont., Can.

J. F. Honsberger, M. D.  
J. M. Livingston, M. D.

## 316      Thirty-Seventh Annual Meeting

Mutual Life of New York, New York, N. Y.	{ E. McP. Armstrong, M. D. H. A. Bancel, M. D. W. M. Bradshaw, M. D. F. H. Carber, M. D. W. S. Gardner, M. D. Lefferts Hutton, M. D. W. C. Huyler, M. D. E. F. Russell, M. D. F. S. Weisse, M. D. R. L. Willis, M. D. E. B. Wilson, M. D.
Mutual Trust Life, Chicago, Ill.	A. A. Willander, M. D.
National Life of U. S. A., Chicago, Ill.	W. A. Jaquith, M. D.
National Life, Montpelier, Vt.	{ G. E. Allen, M. D. A. B. Bisbee, M. D. E. A. Colton, M. D.
New England Mutual Life, Boston, Mass.	{ W. C. Bailey, M. D. D. N. Blakely, M. D. E. W. Dwight, M. D. H. M. Frost, M. D. F. H. McCrudden, M. D.
North American Life Assur., Toronto, Ont., Can.	T. D. Archibald, M. D.
North American Life, Chicago, Ill.	C. B. Irwin, M. D.



## Companies and Representatives 317

New York Life, New York, N. Y.	{ T. W. Bickerton, M. D. R. A. Fraser, M. D. W. H. Hammer, M. D. C. L. Harrison, M. D. A. B. Hobbs, M. D. M. L. King, M. D. E. H. Lines, M. D. Paul Mazzuri, M. D. J. H. North, M. D. O. H. Rogers, M. D. P. E. Tiemann, M. D. H. P. Woley, M. D.
Northwestern Mutual Life, Milwaukee, Wis.	{ J. W. Fisher, M. D. G. A. Harlow, M. D. William Thorndike, M. D. D. E. W. Wenstrand, M. D.
Northwestern National Life, Minneapolis, Minn.	{ H. W. Cook, M. D.
Occidental Life, Los Angeles, Calif.	{ Dudley Fulton, M. D. D. W. Skeel, M. D.
Ohio State Life, Columbus, Ohio	{ C. E. Schilling, M. D.
Pacific Mutual Life, Los Angeles, Calif.	{ W. W. Beckett, M. D.
Pan-American Life, New Orleans, La.	{ Marion Souchon, M. D.
Penn Mutual Life, Philadelphia, Pa.	{ J. P. Chapman, M. D. H. K. Dillard, M. D. D. W. Hoare, M. D. J. U. Hobach, M. D. J. C. Humphreys, M. D. J. P. Hutchinson, M. D. Harry Toulmin, M. D.

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Peoria Life, Peoria, Ill.	{ F. A. Causey, M. D. G. W. Parker, M. D.
Philadelphia Life, Philadelphia, Pa.	{ S. W. Gadd, M. D. J. T. Sheridan, M. D.
Phoenix Mutual Life, Hartford, Conn.	{ A. H. Griswold, M. D. W. D. Morgan, M. D. R. L. Rowley, M. D.
Pilot Life, Greensboro, N. C.	H. F. Starr, M. D.
Provident Mutual Life, Philadelphia, Pa.	{ E. J. Dewees, M. D. Herbert Old, M. D. C. H. Willits, M. D.
Prudential, Newark, N. J.	{ C. T. Brown, M. D. E. G. Dewis, M. D. W. G. Exton, M. D. Paul Fitzgerald, M. D. F. I. Ganot, M. D. G. E. Kanouse, M. D. W. P. Lamb, M. D. L. F. MacKenzie, M. D. J. A. Patton, M. D. J. E. Pollard, M. D. M. K. Smith, M. D.
Public Savings, Indianapolis, Ind.	E. E. Wishard, M. D.
Reliance Life, Pittsburgh, Pa.	{ O. M. Eakins, M. D. W. W. Hobson, M. D. A. A. Wagner, M. D.
Reserve Loan Life, Indianapolis, Ind.	F. L. Truitt, M. D.

## Companies and Representatives 319

Royal Union Life, Des Moines, Iowa	C. M. Whicher, M. D.
Security Mutual Life, Binghamton, N. Y.	W. B. Aten, M. D.
Southland Life, Dallas, Texas	{ J. T. Montgomery, M. D. J. S. Turner, M. D.
Southern States Life, Atlanta, Ga.	C. E. Waits, M. D.
Southwestern Life, Dallas, Texas	Dewitt Smith, M. D.
Standard Life Assur., Montreal, Que., Can.	C. F. Martin, M. D.
State Life, Indianapolis, Ind.	{ C. R. Henry, M. D. J. L. Larway, M. D. C. B. McCulloch, M. D.
State Mutual Life Assur., Worcester, Mass.	{ E. B. Bigelow, M. D. Homer Gage, M. D. C. D. Wheeler, M. D.
Sun Life Assur., Montreal, Que., Can.	{ C. C. Birchard, M. D. J. K. Gordon, M. D. W. F. Hamilton, M. D.
Travelers, Hartford, Conn.	{ J. T. Cabaniss, M. D. W. W. Dinsmore, M. D. P. G. Drake, M. D. L. C. Grau, M. D. F. L. Grosvenor, M. D. G. H. Shaw, M. D. Euen Van Kleeck, M. D. McL. C. Wilson, M. D.

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Union Central Life, Cincinnati, Ohio	{ Charles Maertz, M. D. William Muhlberg, M. D. W. O. Pauli, M. D.
United Life & Accident, Concord, N. H.	R. J. Graves, M. D.
Union Mutual Life, Portland, Maine	A. E. Awde, M. D.
United States Life, New York, N. Y.	{ J. P. Munn, M. D. H. A. Pardee, M. D.
Volunteer State Life, Chattanooga, Tenn.	J. B. Steele, M. D.
West Coast Life, San Francisco, Calif.	M. O. Austin, M. D.
Western States Life, San Francisco, Calif.	H. W. Gibbons, M. D.

## DECEASED MEMBERS.

A. W. Barrows, M. D.	Hartford, Conn.
Charles Bernacki, M. D.	New York, N. Y.
William R. Bross, M. D.	New York, N. Y.
Chauncey R. Burr, M. D.	New York, N. Y.
Robert L. Burrage, M. D.	Newark, N. J.
James Campbell, M. D.	Hartford, Conn.
Frederick W. Chapin, M. D.	Springfield, Mass.
Ferdinand E. Chatard, M. D.	Baltimore, Md.
Thomas C. Craig, M. D.	New York, N. Y.
Edward Curtis, M. D.	New York, N. Y.
Clark W. Davis, M. D.	Cincinnati, Ohio
William B. Davis, M. D.	Cincinnati, Ohio
Charles A. Devendorf, M. D.	Detroit, Mich.
Frank Donaldson, M. D.	Baltimore, Md.
Z. Taylor Emery, M. D.	New York, N. Y.
Thomas A. Foster, M. D.	Portland, Me.
Thomas H. Gage, M. D.	Worcester, Mass.
Donald MacColloch Gedge, M. D.	San Francisco, Cal.
Walter R. Gillette, M. D.	New York, N. Y.
Frank S. Grant, M. D.	New York, N. Y.
Landon Carter Gray, M. D.	New York, N. Y.
Ignatius Haines, M. D.	Boston, Mass.
George C. Hall, M. D.	Richmond, Va.
Edward H. Hamill, M. D.	Newark, N. J.
William W. Hitchcock, M. D.	Los Angeles, Cal.
Edgar Holden, M. D.	Newark, N. J.
John Homans, M. D.	Boston, Mass.
John Homans, 2nd, M. D.	Boston, Mass.
Abel Huntington, M. D.	New York, N. Y.
Arthur Jukes Johnson, M. D.	Toronto, Ont., Can.

John M. Keating, M. D.	Philadelphia, Pa.
William W. Knight, M. D.	Hartford, Conn.
Edward Lambert, M. D.	New York, N. Y.
John B. Lewis, M. D.	Hartford, Conn.
John Mason Little, M. D.	Boston, Mass.
Robert Lanning Lounsberry, M. D.	Binghamton, N. Y.
Henry P. Lyster, M. D.	Detroit, Mich.
Lewis McKnight, M. D.	Milwaukee, Wisc.
Elias J. Marsh, M. D.	Paterson, N. J.
Allison Maxwell, M. D.	Indianapolis, Ind.
Francis D. Merchant, M. D.	New York, N. Y.
William R. Miller, M. D.	Hartford, Conn.
William Natress, M. D.	Toronto, Ont., Can.
Edwin W. Northcott, M. D.	Portland, Me.
Frank K. Paddock, M. D.	Pittsfield, Mass.
William A. Peterson, M. D.	Chicago, Ill.
William E. Porter, M. D.	New York, N. Y.
Albert T. Post, M. D.	New York, N. Y.
James Taggart Priestley, M. D.	Des Moines, Iowa
Oliver P. Rex, M. D.	Philadelphia, Pa.
James F. W. Ross, M. D.	Toronto, Ont., Can.
Gurdon W. Russell, M. D.	Hartford, Conn.
George R. Shepherd, M. D.	Hartford, Conn.
George S. Stebbins, M. D.	Springfield, Mass.
George S. Strathy, M. D.	Toronto, Ont., Can.
Melancthon Storrs, M. D.	Hartford, Conn.
Brandreth Symonds, M. D.	New York, N. Y.
H. Cabell Tabb, M. D.	Richmond, Va.
James Thorburn, M. D.	Toronto, Ont., Can.
James D. Thorburn, M. D.	Toronto, Ont., Can.
Henry Tuck, M. D.	New York, N. Y.
S. Oakley Van der Poel, M. D.	New York, N. Y.

A. L. Vanderwater, M. D.	New York, N. Y.
Clinton D. W. Van Dyck, M. D.	New York, N. Y.
A. C. Ward, M. D.	Newark, N. J.
William P. Watson, M. D.	Newark, N. J.
Joseph H. Webb, M. D.	Waterloo, Ont., Can.
Frank Wells, M. D.	Boston, Mass.
Franklin C. Wells, M. D.	New York, N. Y.
George W. Wells, M. D.	New York, N. Y.
A. H. Whitridge, M. D.	Baltimore, Md.
George Wilkins, M. D.	Montreal, Que., Can.
G. S. Winston, M. D.	New York, N. Y.
Albert Wood, M. D.	Worcester, Mass.
Green V. Woollen, M. D.	Indianapolis, Ind.
Joseph C. Young, M. D.	Newark, N. J.

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